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CELIAC SYNDROME

I: DETERMINATION OF FAT IN FECES; RELIABILITY OF TWO CHEMICAL METHODS AND OF MICROSCOPIC ESTIMATE; EXCRETION OF FECES AND OF FECAL FAT IN NORMAL CHILDREN

DOROTHY H. ANDERSEN, M.D.

NEW YORK

teatorrhea is a common finding in infants children with chronic indigestion and is cially frequent in patients who present the ac syndrome. The naked eye diagnosis of y stool is unreliable, however, for the large foamy stools of the patient with celiac dis- may or may not contain a relative excess at, while the fatty stools of the patient with creatic deficiency may appear normal. It is herapeutic as well as academic interest to dis- uish the cases in which there are an excess fecal fat and consequent gross loss of fat- ble vitamins from those in which utilization at is within normal limits. Fat is normally ent in feces, even during starvation [1]; steator- a is correctly defined as the presence of an ess of fat in the stools. Proof of the presence absence of steatorrhea therefore requires a ntitative method for determination of fecal fat an adequate series of control determinations comparable subjects. Since fecal excretion of is affected by diet, the latter should also be trolled. The primary objects of the present dy are the evaluation of several methods for ermination of fecal fat and the establishment control figures by analyses of the stools of rmal infants and children while they are re- ving a diet customary for their age at this time place.

The multiplicity of methods and modifications methods which have been used in analysis of es for fat indicates that none is entirely satis- tory. In the present series of investigations method was required which could be used in a ge series of cases without making overwhelm- demands on the hospital wards and laboratory which would yet have a high degree of re- bility. The earlier observations reported here were made by a chemical method based on a procedure employed by Sperry [2] and modified by him in unpublished experiments [3]; in most clinical laboratories, however, some modification of the Fowweather method [4] is used. A comparison of the results of analysis by these two methods of a series of samples of normal and of pathologic feces seemed of importance in evaluating these methods and in providing a basis for comparison of data obtained from various laboratories. The Fowweather method with the use of wet material was selected in preference to the modifications of this method in which dried material is used, such as the procedure of Tidwell and Holt,[5] on the assumption that the former method is more widely used. Methods involving collection of feces in alcohol or other solvents or preservatives were not considered, since these methods prevent measurement of total fecal excretion per day, measurement of water content and analysis for substances other than fat.

Since both methods were found to be too time-consuming for routine clinical use, a microscopic examination of fresh feces stained with sudan IV was made for comparison with the results of chemical analysis, in the hope that this simple test might be used to screen out the patients without steatorrhea.

SUBJECTS AND METHODS

Selection of Patients.—The control patients were so selected that they were all of normal weight and normal physical development for their age and were

From the Babies Hospital and the Departments of iatrics and Pathology, Columbia University Col- of Physicians and Surgeons.

1. Sperry, W. M., and Bloor, W. R.: Fat Excre- : II. The Quantitative Relations of the Fecal Lipoids, Biol. Chem. **60**:261-286, 1924.

2. Sperry, W. M.: Lipid Excretion: III. Further Studies of the Quantitative Relations of the Fecal Lipids, J. Biol. Chem. **68**:357-383, 1926.

3. Sperry, W. M.: Personal communication to the author.

4. Fowweather, F. S.: The Determination of the Amount and the Composition of the Fat of Feces: I. Investigation of a "Wet" Method and Comparison with the "Dry" Method, Brit. J. Exper. Path. **7**:7-14, 1926.

5. Tidwell, H. C., and Holt, L. E., Jr.: The Estimation of the Total Lipids and the Lipid Partition in Feces, J. Biol. Chem. **112**:605-613, 1936.

eating well. No subjects were included who were known to have had diarrhea; all were afebrile at the time of the observation and had been afebrile for at least four days previously. In most instances the diagnosis in the hospital was one of the following conditions: acute pneumonia, (patient recovered); cervical adenitis, (recovered); mental deficiency; lesion of the central nervous system; benign tumor (i. e., lipoma or hemangioma); congenital or rheumatic heart disease without cyanosis or decompensation; malnutrition because of inadequate food (recovered), and no abnormal condition.

Material for the comparative studies of various methods of determination of fat was obtained from various patients, some of whom presented the celiac syndrome, with or without steatorrhea. For the purposes of the present paper the data derived from these sources are considered only in respect to the accuracy of the method used. Clinical correlations of these and other observations will be reported subsequently.

Diet.—The control patients and the majority of the others studied were receiving the diet normal for their age during the period of collection and for at least one day prior to the beginning of the collection of fecal material. This diet is referred to in the present paper as the "ward diet." Since the diet given infants varies greatly in different countries, in different parts of the same country and even in different hospitals in the same city, the ward diet is described in some detail. Some of the patients used for the study of methods were receiving modified diets.

No effort was made to measure the exact amount of food eaten. No patient was accepted as a control, however, who was not considered to be eating well and therefore receiving approximately the estimated amount of fat provided in the menus. Approximate menus were as follows:

Age 2 to 6 Months: Formula: Evaporated milk, 1 part; water, 2 parts, and sucrose, 5 per cent.

Breakfast: Three ounces (90 cc.) of orange juice, ½ cup of cereal, 1 teaspoon of sucrose, 6 ounces (190 cc.) of the formula and 1 egg (after the third month).

Lunch: One-fourth cup of puréed vegetable, ¼ cup of puréed fruit and 6 ounces of the formula.

Supper: One-half cup of cereal, 1 teaspoon of sucrose, ¼ cup of vegetable and 6 ounces of the formula.

10 p. m.: Six ounces of the formula.

The total number of calories is 861; the carbohydrate content, 120.8 Gm., or 56 per cent of the calory value; the protein content, 28.8 Gm. or 13 per cent, and the fat content, 29 Gm., or 30 per cent.

Age 6 to 12 Months: Formula: At about the age of 8 months the transition is made from the formula just given to pasteurized whole cow's milk.

Breakfast: As before, with the addition of 1 piece of zwieback.

Lunch: One ounce (28.4 Gm.) of meat (usually scraped beef), ½ cup of potato, ¼ cup of puréed vegetable, ¼ cup of puréed fruit and 6 ounces of the formula.

Supper: One-half cup of cereal, 1 teaspoon of sucrose, ¼ cup of vegetable, ¼ cup of dessert (pudding or gelatin), 1 piece of zwieback and 6 ounces of the formula.

The total number of calories is 1,285; the carbohydrate content, 148 Gm., or 46 per cent of the calory value; the protein content, 48.6 Gm., or 15 per cent, and the fat content, 55.4 Gm., or 39 per cent.

Age 1 to 2 Years: Breakfast: Four ounces (120 cc.) of orange juice, ½ cup of cereal, 1 teaspoon of sucrose, 1 egg, ½ slice of toast, ½ pat of butter 6 ounces of milk.

Lunch: One ounce (28.4 Gm.) of meat, ⅓ cup p or a substitute, ½ cup of vegetable, ½ cup of and 6 ounces of milk.

Supper: One-half cup of combined carbohydrate protein (i. e., spaghetti with meat sauce), ⅓ c vegetable, ½ slice of toast, ½ pat of butter, ½ c dessert and 6 ounces of milk.

Supplement: Six ounces of milk additional.

The total number of calories is 1,480; the carb drate content, 147 Gm., or 40 per cent, of the c value; the protein content, 60 Gm., or 16 per cent, an fat content, 75 Gm., or 44 per cent.

Age 2 to 6 Years: Breakfast: Six ounces orange juice, ½ cup of cereal, 1 ounce (30 cc. cream, 1 teaspoon of sugar, 1 egg, ½ slice of with butter and 6 ounces of milk.

Lunch: Three ounces (85 Gm.) of meat, fish chicken; 3½ ounces (127.5 Gm.) of potato, 2½ o (70 Gm.) of cooked vegetable, ½ ounce (14 Gm. raw vegetable, ½ slice of bread with butter, ra cooked fruit and 6 ounces of milk.

Supper: One-half cup of combined carbohydrate protein, 2½ ounces of cooked vegetable, ½ slic bread with butter, ½ cup of dessert (pudding, cus ice cream or cake) and 6 ounces of milk.

The total number of calories is 1,715; the carbohyd content, 190 Gm., or 45 per cent, of the calory va the protein value, 71 Gm., or 16 per cent, and the value, 75 Gm., or 39 per cent.[6]

The calculations were based on the sample m just given; the distribution of calories varied but l from day to day.

PROCEDURE

Collection of Stools.—The patients began to rec the chosen diet one or more days before collection begun. The preliminary period was made short bec many patients with the celiac syndrome tolerate a nor diet poorly. For infants having one or more stools d one day is probably a sufficiently long prelimin period. The stools were collected for seventy-f hours in most instances, but this period was occas ally shortened to forty-eight hours for patients w diarrhea or lengthened to ninety-six hours when stools were few or small. Each diaper was placed a paper bag, or the stool was put in a closed contai and placed at once in a refrigerator. Once a day, sometimes at the close of the collection period, diapers were carefully scraped with a blunt knife a tongue depressor and the material collected i covered tared beaker. There was unavoidable loss feces on the diapers, but aside from water this appea to represent only a small fraction of the material exc for patients with watery diarrhea. This method w used in preference to the somewhat more accurate o of extracting the entire diapers or using a metaboli bed, since it was not feasible to apply these more co plicated methods routinely to the study of large ser of patients.

Analysis of Stools.—1. Microscopic Examination: fresh sample of stool was examined. In most instan a stool which was brought to the laboratory at a time during the day was used. A small amount of st usually a mass 1 to 2 mm. in diameter, was placed a glass slide. A few drops of a saturated alcoho solution of sudan IV was added and the mixture stir

6. Miss Emma Mike, dietitian at Babies Hospi provided these data.

oughly with an applicator stick. Since the alcohol orated in part during this process, a drop of 0.7 cent solution of sodium chloride was stirred in and ver glass pressed over the preparation, the excess being wiped from the margins. The mixture was examined with low and with medium magnifica-

The amount of fat was estimated and recorded llows: 0 to +, two droplets of fat or less per low er field; +, not more than four to five droplets; and ++, intermediate but abnormal amounts, ++++, droplets of fat or fatty acid crystals con- ting about half the visible material. Independent mates by several observers agreed approximately; is, they fell within two adjacent classifications. various estimates of a sample have sometimes +++ and ++++ but never ++ and ++++.

Determination of Wet Weight, Water Content Dry Weight: After the entire collection had been ed in a tared beaker, it was weighed. This pro- re gave the total weight for the collection period, from it the average daily wet weight could be ulated. The contents of the beaker were then roughly stirred with a glass rod. About 3 to 5 Gm. then transferred to a tared 30 cc. beaker and kly weighed to the nearest milligram. This beaker left in a drying oven at 100 C. for forty-eight rs and then placed in a desiccator over anhydrous ium chloride for cooling. It was weighed twenty- and forty-eight hours later. If these weighings not check to 0.2 mg. the beaker was weighed daily il constant weight was attained, but additional ghings were rarely necessary. The values for the er content of the stool and the dried weight of the l per day could then be calculated.

. Analysis of fat: The Fowweather method has been viously described.[4] The Sperry method will be ex- ined in detail, because it has not been recorded in entirety.

. Sperry Method: This is a standard chemical hod, which has been attributed to Sperry because technic of extraction is used.[7] An aliquot of 30 to Gm. of feces is placed in a tared 250 cc. beaker weighed quickly to the nearest tenth of a gram. ncentrated sulfuric acid is then added; in the earlier periments 3 drops of acid was stirred thoroughly the material and the reaction tested with blue nus paper; if the litmus paper did not become a dis- t red, further acid was added until the reaction was d. In later experiments 3 or 10 drops of acid was ded. Powdered anhydrous sodium sulfate approxi- tely equal in volume to the sample of stool is stirred and if necessary more is added until the mixture the consistency of brown sugar. The mixture is n transferred to a muslin bag with a length of about cm. and a circumference of 6 cm. at the top and cm. at the bottom. The bag is equipped with four pes, by which it can be suspended in a 1 pint (0.5 er) percolator, as in the type of extraction apparatus scribed by Sperry.[2] The percolator is fitted by a bber stopper into a 1 liter, round-bottomed flask. eat is provided by a 40 watt electric bulb enclosed a can under the flask. The temperature in the flask kept between 45 and 50 C. by this means. The ex- action solvent is vaporized and passes up along the les of the bag. It is condensed by a coil of copper bing suspended over the bag and the mouth of the rcolator. The tubing is ¼ inch (0.64 cm.) in diam- r and is coiled in eight or more spirals, most of which are below the rim of the percolator. Cold water runs continuously through this coil.

After the bag has been transferred to the percolator, the beaker and the rod are rinsed three times with ethyl ether into the bag, and about 100 cc. more of ether is added by being poured through the bag. The heat is then applied and extraction continued for three to five hours. The whole is allowed to cool, usually overnight. The bag is then emptied into a large mortar and the material thoroughly ground; then it is carefully returned to the bag and again extracted until a total extraction period of eight hours has elapsed. More ether is added as required, but the loss of ether is usually slight. After cooling, the contents of the flask are transferred with washings to a 500 cc., pear-shaped separatory funnel. If crystals of sodium sulfate have accumulated in the flask, these are dissolved in water with gentle heating. After cooling, this washing is added to the contents of the funnel and the flask washed once more with ether.

The ether extract in the first separatory funnel (the "neutral fat" funnel) is then washed three times with distilled water, six to twenty-four hours being allowed for the layers to separate after each washing. The washings are discarded. The ether layer is then extracted three times with approximately 75 cc. of fifth-normal solution of sodium hydroxide, the alkaline extracts being transferred successively to a second funnel, the "fatty acid" funnel. The ether extract in the neutral fat funnel is then washed three times with distilled water; the washings are also transferred to the second funnel. After each extraction or washing a period of six to twenty-four hours is required to allow separation of the layers; for some of the specimens from patients with the celiac syndrome this period is greatly prolonged. The ether extract in the first funnel is finally transferred with careful ether washings to a tared 100 cc. beaker. The ether is allowed to evaporate at room temperature. The beaker is left in an incubator at 37 C. until all visible ether and water have disappeared and the weight is constant to within 0.5 mg.; in the case of neutral fat this goal is usually attained at the second or third weighing. This fraction consists of esters and unsaponifiable material and gives the figure for neutral fat.

The first alkaline extract, which has been transferred from the neutral fat funnel to the fatty acid funnel, is acidified with an excess of normal solution of sulfuric acid; 50 to 100 cc. of ether is added and the funnel is shaken well and allowed to stand. After the layers have been separated, the aqueous layer is tested with litmus to make certain of its acidity and is then discarded. Some volatile fatty acid may be lost with it. The second alkaline extract is then added, and the process is repeated until the lipids of all the alkaline extracts and washings have been collected in the ether layer of the "fatty acid" funnel. The excess acid and salt are removed by three washings with distilled water; the ether layer is then dried and weighed in the same manner as the neutral fat. This fraction contains the fatty acids from soaps as well as free fatty acids and gives the figure for total fatty acids.

Calculations are made as follows:

$$\text{Wet weight of aliquot} \times \frac{\text{Per cent of solids in stools}}{100} = \text{dry weight of aliquot.}$$

$$\frac{\text{(Gm. of neutral fat)}}{\text{Dry weight of aliquot}} \times 100 = \text{A, or neutral fat, per cent of solids.}$$

$$\frac{\text{(Gm. of fatty acids)}}{\text{Dry weight of aliquot}} \times 100 = \text{B, or fatty acids, per cent of solids.}$$

7. Sperry (footnotes 2 and 3).

A + B = C, or total fat, per cent of solids.

C × (grams of solid excreted per day) = total fat excreted per day.

The amount of neutral fat and of fatty acids excreted per day is calculated similarly.

B. Fowweather method: This method was employed as described by the originator of the method.[4] Several details were modified: (1) Three grams ± 0.1 Gm. of stool was used in each cylinder, and (2) three or more extractions with ether were made, until the final extraction was colorless. The titration factor used in calculation of fatty acids was 1 cc. of tenth-normal sodium alcoholate = 0.0283 Gm. of fatty acid rather than the 0.0268 suggested by Harrison[8] and used by Tidwell and his associates.[5] The reason for this choice is given later.

OBSERVATIONS

Reliability as Measured by Likeness of Duplicate Determinations; Comparison of Fowweather and Sperry Methods.—Duplicate determinations were made on 60 samples of feces by the Fowweather method and on 10 samples by the Sperry method. The feces were obtained from infants and children who were receiving various diets which contained meat, milk and vegetables. The patients for whom the Fowweather method was used included controls without nutritional difficulty, patients presenting various forms of the celiac syndrome and a few patients who had steatorrhea for other reasons, such as congenital atresia of the bile ducts. Since the amount of material required for duplicate determinations by the Sperry method is greater than that usually provided by a three day collection from normal infants, the determinations by this method were made on material from patients who excreted an abnormally large amount of feces; 5 of patients had a diagnosis of pancreatic deficie 1 of celiac disease with steatorrhea and 4 of c syndrome without steatorrhea.

It will be seen from table 1 that the differe between duplicate determinations made by Sperry method is of the order of one-third by the Fowweather method. The error in latter method is of the same order for the var groups of patients with the exception of a gr of 6 patients with pancreatic deficiency, for wh the error was less but was still greater than the Sperry method. This statement applies to various fractions not recorded in the table as as to the total fat. Study of the figures give table 1 suggests that the error with the F weather method would probably be lessened

TABLE 1.—*Likeness of Duplicate Determinations: Comparison of Fowweather and Sperry Methods*

Fraction	Method	Group	No. of Subjects	Mean, Gm./Day	Mean Difference, Gm./Day	Mean per Ce Differe
Total fat	Fowweather	Controls	14	1.52	0.11	7.5
		Diarrhea, low fat	11	1.93	0.13	7.1
		Diarrhea, high fat	21	6.83	0.49	6.8
		Pancreatic deficiency	6	16.84	0.56	3.7
		Atresia of bile ducts	5	7.83	0.74	6.8
		Series	60	5.52	0.35	7.1
	Sperry	Series	10	8.70	0.16	2.0
Neutral fat	Fowweather	Series	60	1.09	0.11	12.5
	Sperry	Series	10	2.00	0.08	4.8
Fatty acids plus soaps	Fowweather	Series	60	4.40	0.34	9.3
	Sperry	Series	10	6.42	0.14	3.0
Soap	Fowweather	Series	60	1.30	0.35	32.7
Free fatty acids	Fowweather	Series	60	3.15	0.16	7.7

$$\text{Mean } \Sigma \frac{\frac{A+B}{2}}{N}\text{; mean difference } \Sigma \frac{\text{Difference between A and B}}{N}$$

$$\text{Mean per cent difference } \Sigma \frac{\frac{\text{Difference between A and B} \div (A+B) \times 100}{2}}{N}$$

A and B = figures obtained in duplicate determinations on the same sample and also (in this table) by the same method.

Comparison of Parallel Determinations Made by the Two Methods on the Same Samples.—A single determination by the Sperry method was compared with the mean of duplicate determinations by the Fowweather method on 61 samples of feces. In these observations the aliquot used for the Sperry method was acidified by addition of 3 or more drops of concentrated sulfuric acid until the material showed an acid reaction to litmus paper. Analysis of these data (table 2 [A]) shows that the mean figure for total fat for the series is distinctly higher when calculated by the Fowweather than by the Sperry method. This result is surprising, since there appears to be greater error in extraction and greater loss in transfer with the Fowweather method. (

8. Harrison, G. A.: On the Differential Estimation of Fat in Feces, Brit. J. Exper. Path. **6**:139-146, 1925.

ionation this difference is found to be due difference in the amount of fatty acid plus , while the mean figure for the neutral fat ion is somewhat higher with the Sperry hod. This raises the questions: What causes difference and which figure is correct?

everal possible reasons for the discrepancy be considered. 1. When material for the weather method is selected, the larger pieces ndigested material, especially of vegetables, usually avoided. Thus a significant error be introduced, which is not present with Sperry method because of its large aliquot. 2. concentrated acid used with the Fowweather hod may free lipids which are bound to teins or carbohydrates and which are not d by the smaller amount of acid used with Sperry method. 3. Acids other than fatty s may be included in the Fowweather titra- . 4. The factor used in calculating fatty s by the Fowweather method may be inaccu- . 5. The heat used in drying the final product he Sperry method may drive off the more tile fatty acids. It seems unlikely that these

LE 2.—*Comparison of Results of Analysis of Feces or Fat by Fowweather and Sperry Methods on Same Samples: Effect of Amount of Acid Added in Sperry Method*

Experiment	No. of Subjects	Fowweather, Gm./Day	Sperry (Acid to Litmus) Gm./Day	Difference Between Means	Mean Difference
Total fat.....	61	5.64*	5.00	0.64	1.03
Neutral fat.....	61	1.05	1.15	0.10	0.41
Total fatty acid...	57	4.89	4.05	0.84	1.14
		10 Drops Acid			
Total fat.....	20	6.20	5.38	0.82	0.35
	19	4.79	4.77	0.02	0.19
	1	29.52	16.90		
		3 Drops Acid			
Total fat.....	20	6.20	4.43	1.77	0.91
	19	4.79	4.00	0.79	0.52
	1	29.52	12.75		

The figures for the Fowweather method represent $\Sigma \frac{A+B}{2}$ ns of the mean of duplicate determinations, i. e. $\frac{}{N}$

mean difference $\Sigma \frac{\text{Difference between results of Sperry and Fowweather methods on same samples}}{N}$

present in sufficient quantity to account for discrepancy, however. 6. The small volume acid used with the Sperry method may be sufficiently mixed into the mass of material and nce may leave fatty acids bound as soap. These uld be incompletely extracted.

Inspection of the individual data showed that a rule the greatest discrepancies in the figures tained by the two methods were for those mples having a high fat content, especially a gh soap content. This observation suggested at the factor last mentioned might at least con- bute largely to the discrepancy. A second series of determinations was therefore made in which the mean of duplicate determinations by the Fowweather method was compared with the results of 2 determinations by the Sperry method, in one of which 10 drops of concentrated acid was used, and in the other 3 drops. The three day collections of feces used were of necessity obtained from patients having more than the normal volume of feces. Twenty such collections were analyzed in this way; the results are given in table 2 (B and C). It is evident that when the Fowweather is compared with the Sperry method using 10 drops of acid close checks were obtained in all instances except 1, in which the total amount of fat, especially of soap, was excessive and in which, by unlucky chance, an unusually large aliquot, 64 Gm., was used. Since this experience the precaution has been taken of using smaller aliquots, about 30 to 40 Gm., and 20 drops of acid when microscopic examination has shown an unsually large amount of fat and of using 10 drops of acid routinely in all other determinations. It is also obvious from the data that 3 drops of acid is insufficient in many instances. Since many of the determinations recorded in this and subsequent papers were made before this source of error was discovered, it is of importance to have the means of evaluating the probable amount of error introduced in this way. It is also interesting to observe that for this series the mean difference between duplicate determinations by the Fowweather method of total fatty acids in 0.18 Gm. per day, with a mean percentage difference of 3.25, a figure much lower than in the earlier experiment. This probably reflects the greater fat content of the feces examined, as was suggested in the preceding section.

The evidence presented in these two sections leads to the conclusion that a single determination of total fat by the Sperry method with the use of 10 drops of acid is more reliable than a single determination by the Fowweather method and about equal in reliability to the mean of duplicate determinations by the latter method. The smaller quantity of materials required for the Fowweather method makes feasible duplicate determinations, which are often impossible to carry out with the Sperry method. The labor required is less with the Sperry method.

Sources of Error with the Fowweather Method.—The wide variation in duplicate determinations leads to examination of sources of error in the various steps of this method.

A. Selection of the Aliquot: In using aliquots as small as 3 Gm. of a far from homogeneous material an error is necessarily introduced. Children of the toddler age often have large frag-

ments of vegetable in the stools. This is especially true of patients with chronic indigestion, with whom pieces of vegetable may appear to form a large fraction of the feces. In selecting aliquots for analysis these pieces of formed material are usually avoided, and therefore there is an abnormally high estimate of the daily excretion of fat and also a difference between duplicate determinations. This result may be avoided by drying and grinding the feces, as has been done by Tidwell and his associates and by many others; it is avoided with the Sperry method by the use of large aliquots.

B. Extraction: In the original extraction of the feces it is found that some samples, especially those containing mucus, are not well suspended in the alcohol-acid-ether mixture, in spite of vigorous shaking. The record shows that these samples usually give poor checks, as might be expected. Difficulty in breaking up the emulsion is also sometimes experienced. In this laboratory the problem has been solved by permitting prolonged standing, with occasional rotation of the cylinder. The difficulty may also be solved by centrifugation of the emulsion if appropriate tubes are used in the original extraction.

C. Loss in Tranfer: In transfer of the supernatant ether layer by blowing into the beaker there is slight loss because of spraying. With care this becomes imperceptible. No method of measuring this loss has been devised.

In the filtration of the material and the associated transfer to a second beaker there is a slight loss. Any attempt to measure this loss by weighing the extracts before and after transfer is futile; since the solvent is ethyl ether in the first instance and purified benzine in the second, some loss is to be expected.

The loss during the third transfer—that of the weighed purified benzine extract into the flask used for titration—together with the accuracy of the titration, can be evaluated by comparison of the amounts of fatty acid obtained per gram of dried purified benzine extract. These calculations made on the series of 61 determinations recorded in tables 1 and 2 give a mean difference between duplicate determinations of 2.7 per cent, a figure which accounts for about one third of the total error found in this series.

D. Titration Factor: In the present experiments the factor 1 cc. of tenth-normal sodium hydroxide = 0.0283 Gm. of fatty acid was used. This factor was recommended by Fowweather, on the assumption that fecal fatty acids are chiefly stearic acid. The factor 0.0268 was subsequently recommended by Harrison,[8] who obtained it by analysis of a pooled collection of fatty acids from the feces of children of various ages up to 12 years. Tidwell and his associates [5] also used latter factor. It seemed possible that the di ences in the diet of infants and of young chil and the possibility that patients with imp digestion or absorption of fat may excrete tain fatty acids selectively might result in v tions in the mean molecular weight of the fatty acids. To evaluate the error so introd the titration factor was determined for the acids obtained from the feces of three group patients: (1) patients having daily fat excr within the normal range, (2) patients with disease and steatorrhea and (3) patients pancreatic deficiency. All the patients were ceiving a mixed diet, which included cow's m meat and vegetables. They ranged in age f 7½ months to 5 years. The fatty acids obtained from the feces by extraction with e by the Sperry method and were then taken u purified benzine and filtered, dried and reweig in order that their preparation might be c parable to that with the Fowweather meth In 19 determinations the material recovered extraction with purified benzine amounted t mean figure of 94.8 per cent of the fatty ac extracted with ether. The titration factors tained from these samples are given in table

TABLE 3.—*Titration Factor of Fecal Fatty Acids Patients Having Normal Excretion of Fat, Celiac Disease with Steatorrhea and Pancreatic Steatorrhea*

Group	No. of Subjects	Age Range	Factor: 1 Cc. of N/10 NaOH = Grams of Fatty Acid		
			Mean	Range	S.D.
Normal fat excretion	8	12 mo.-3½ yr.	0.0285	0.0270-0.0317	0.00
Celiac steatorrhea	5	7½ mo.-18 mo.	0.0282	0.0275-0.0291	0.00
Pancreatic steatorrhea	6	10 mo.-5 yr.	0.0290	0.0276-0.0309	0.00
Total series	19	.....	0.0286	.....	0.00

It is evident that there is no significant differe among the factors obtained in the various grou of cases, and the figure is found to approxim that of Fowweather more closely than that Harrison, with a surprisingly wide standa deviation. No factor is accurate unless it obtained for the individual sample for which is used.

The titration factor was also obtained for fatty acids of a series of common food fats. T mutton, beef and pork fat were obtained by tr ing out fresh fat. The milk fat was prepared centrifugation of evaporated milk. The corn and the olive oil were used as purchased. T fats were hydrolyzed as follows: Twenty gra of fat, 55 cc. of water, 12 cc. of molar solution ammonium chloride, 4 cc. of molar solution

onia, 4 cc. of 2 per cent solution of calcium ride, 4 cc. of filtered 2 per cent egg albumin Gm. of pancreatin were thoroughly shaken incubated for twenty-four hours at 37.5 C. cond gram of pancreatin was then added he flask shaken well and again incubated for ty-four hours. The mixture was placed in paratory funnel and acidified, and the fatty separated by the Sperry method. The rs were obtained after reextraction with ied benzine. The following factors are the t of duplicate determinations: evaporated 0.0260 and 0.0260; mutton fat, 0.0273 and 75; breast milk, 0.0284 and 0.0283; beef fat, 89 and 0.0293; corn oil, 0.0297 and 0.0312; fat, 0.0298 and 0.0302; olive oil, 0.0327 and 14. The fats of a mixed diet including milk meat may be expected to show a variation in this range, and the factor 0.0283 appears onable. The differences between the factors ined by various authors are probably the lt of differences in diet. It may be expected the mean molecular weight of the fecal fatty s reflects that of fed fat in the same manner hich the iodine number is reflected.[9]

. Cumulative Error in Determination of Soap ctions: The difference between duplicate rminations is far greater with soap than the other fractions. Moreover, a negative e was obtained in 1 or both determinations 7 of 60 samples. These two observations doubt on the value of determining soap by method. In considering possible causes of wide error it should be recalled that the unt of soap is not determined directly but is ulated by subtraction of the unacidified from acidified aliquot. The error in the value for p therefore represents the accumulated error he entire procedure of extraction and transfer both aliquots. Inspection of the data shows t the error is of the same order in the samples taining high and low fat and that the higher re is more than twice the lower in 14 duplie determinations, or 23 per cent of the samples. is doubtful whether determination of soap by s method is of value in the study of individual mples, though it may be reliable in a series cases if sufficient data are available for statisal analysis.

Sources of Error with the Sperry Method.—Selection of the Aliquot: The aliquot is fficiently large to prevent the introduction of y large error in selection.

B. Acidification: It has already been shown that inadequate acidification will lead to failure to extract soaps. Ten drops of concentrated sulfuric acid proved adequate for all samples except 1 with unusually high fat content, in which an unusually large aliquot was used. It is suggested that smaller aliquots and more acid be used when microscopic examination shows a ++++ amount of fat. If this precaution is taken, no error should be introduced with this step.

C. Extraction: The extraction of material which is soluble in ether approaches completeness. Further extraction after the customary eight hours yields imperceptible amounts of fat.[3]

D. Transfer: There are three possible sources of error in the handling of the separatory funnels. The first is the usual difficulty with leaking funnels, which can be reduced by placing a clean beaker under each funnel. The second is loss in the process of shaking the funnel, which can be avoided by care in procedure. The third is the possibility of failure adequately to acidify the alkaline extract in the second funnel. This source of error can be minimized by testing the aqueous layer in the second funnel with litmus before discarding it.

Significance of the Various Fractions.—Neutral Fat: In the present experiments both glycerides and unsaponifiable material are included in the neutral fat fraction. It has been shown that with fat-free diets and during starvation there is still fecal excretion of fat. Moreover, fecal excretion of fat is only slightly increased above the fasting level when adequate diets containing usual food fats are given. In normal subjects receiving either fat-free or normal diets a considerable proportion of the fecal fat, about 10 per cent in infants,[9] is unsaponifiable material. Sperry and Angevine[10] have presented evidence in favor of the view that this endogenous fecal fat is secreted into the intestine. Calculations based on data supplied by Holt and his associates[9] indicate that for infants of 4 to 6 months receiving a diet low in fat the unsaponifiable fecal lipids amount to about 0.2 Gm. per day. It is possible that older infants receiving a mixed diet also excrete a small amount of unsaponifiable material derived from food. According to my analyses, as presented in table 5, the total daily excretion of neutral fat (i. e., glycerides and unsaponifiable material together) does not greatly exceed Holt's figure for unsaponifiable fat alone. No evidence has been

9. Holt, L. E., Jr.; Tidwell, H. C.; Kirk, C. M.; oss, D. M., and Neale, S.: Studies in Fat Metabon: I. Fat Absorption in Normal Infants, J. Pediat. 27-480, 1935.

10. Sperry, W. M., and Angevine, R. W.: Lipid Excretion: IX. The Secretion of Lipids into the Intestine, J. Biol. Chem. **96**:769-786, 1932.

encountered which casts doubt on the statement of Tidwell and Holt: "Only in pathologic states and with certain very poorly absorbed fats does the true neutral fat constitute a considerable proportion of the feces." [5] Since the unsaponifiable fraction and hence the neutral fat fraction are constantly present and remain above a certain minimum level, they may form a large proportion of fecal fat if absorption of glycerides and fatty acids is good and a small proportion if absorption of dietary fat is poor. The absolute figures for daily excretion of neutral fat are therefore more informative than the relative figures.

Experience has shown that patients with pancreatic deficiency and absence of pancreatic lipase from the duodenal juice may have fatty stools which on analysis give low values for neutral fat. Oral administration of pancreatin will reduce the amount of fat excreted per day without necessarily altering the ratio of neutral fat to

As is shown in table 4, the total fat was sim in the 2 samples; but in the second sample proportion of neutral fat was much decrea varying amounts up to one third of the amo of neutral fat found in the first aliquot w split and in most instances the proportion total fat present as neutral fat was decreased normal levels. Since this loss took place a low temperature over a period of forty-e hours, it is probable that significant splitting neutral fat had previously taken place in colon at body temperature, in the diaper at r temperature (in much shorter periods of tim and in the refrigerator while the collection being made. It is probable that most, if not of the excess of undigested fat excreted patients with pancreatic deficiency is in the fo of neutral fat as it enters the colon. It is evident that failure to discover an excess neutral fat by analysis of the feces does not prove the diagnosis. High values for neu

TABLE 4.—*Decrease in Proportion of Neutral Fat in Feces of Patients with Pancreatic Steatorrhea Resu from Letting the Specimen Stand Forty-Eight Hours Under Refrigeration* *

Patient	Age	Diet †	Total Fat, Gm./Day		Neutral Fat, % of Fat		Neutral Fat, Gm./Day		Difference	
			A	B	A	B	A	B	Gm.	%
C. J.	2 yr.	III	30.5	30.4	36.8	28.3	11.23	8.60	—2.57	—2
		IV	28.1	30.7	25.8	16.4	7.25	5.03	—2.22	—3
		V	19.9	22.1	37.8	22.7	7.53	5.01	—2.52	—3
		VI	23.3	23.6	34.0	24.7	7.92	5.82	—2.10	—3
R. H.	8 mo.	I	8.95	8.02	33.0	27.2	2.96	2.18	—0.98	—3
		II (low fat)	1.34	1.45	40.8	32.5	0.54	0.47	—0.07	—1
M. G.	2¾ yr.	...	12.0	13.8	19.2	12.1	2.29	1.67	—0.62	—2

* A, analysis begun at end of three day collection; B, analysis of same specimen after standing forty-eight hours at 6 to
† The composition of the several diets referred to is irrelevant to the subject of the present paper. The diets will be
described in a later report.

total fat. Close checks in duplicate determinations by each of the methods used and comparison of the results with the two methods give evidence that the low figures sometimes obtained represent accurately the neutral fat content of the sample analyzed. The possibility must be considered that the food fat is split under these circumstances in some manner not related to the normal digestive process—perhaps by bacterial action in the colon, or even in the collection beaker. Although in these studies the feces have been kept under refrigeration at 6 to 7 C. while collections were being made, the question of saponification in vitro cannot be dismissed without specific inquiry. In order to test this hypothesis, duplicate determinations were made on a series of three day collections of feces from patients with congenital pancreatic deficiency. The first aliquot was acidified and extracted on the morning on which the collection was completed; the second aliquot was left covered in the refrigerator for forty-eight hours before analysis. All analyses were made by the Sperry method.

fat, on the other hand, are significant. T may be misleading if the patient has been gi mineral oil without the knowledge of the inve gator.

In some of the control samples a low fig for fecal fat was associated with a high prop tion of neutral fat. It seems reasonable to int pret this circumstance as the result of good a sorption of fat, leaving chiefly unsaponifiable fat the feces. An increase in the amount of neutral may be encountered in the feces of normal infan fed certain poorly digested fats [9] or in patients w diarrhea from any cause; yet the daily excreti of neutral fat may be normal for patients w pancreatic deficiency who are receiving di low in fat or are eating poorly. If factors diet, medication and technic are taken into co sideration, the estimation of neutral fat in fe is of occasional, but limited, clinical value.

Total Fatty Acids and Soaps: The fatty ac excreted as soaps probably reflect the p_H of t stools and the amount of mineral cations prese as has been suggested by previous authors.

fficult to explain in any other way the pres- of large amounts of soap in the stools of nts with pancreatic deficiency, in whom difficulty is primarily one of splitting fat. reduction of the soaps in patients with true c disease during acidotic crises[11] may also nterpreted on this basis, though here the tion is still more complicated. In view of greater ease and accuracy of other methods etermining the p_H and the close correlation een the p_H and the size of the soap fraction, rmination of the latter has limited value also. can determine the total fatty acids with fair racy; they account for the major portion of ss fat in most varieties of steatorrhea. The ificance of the fatty acids is in their relation he proportion of neutral fat present, which already been discussed.

otal Fat: The daily fecal excretion of total varies within a relatively narrow range in infants under 6 months this proportion is higher than in older infants.

Wet Weight, Dried Weight and Water Content of Stool: Under the conditions of my observations the maximal figure for daily excretion of feces in terms of either wet weight or dry weight is within twice the mean for each age group. This means that for children under the age of 6 years excretion of more than 50 Gm. wet weight or 13 Gm. dried weight is beyond the limits of normal. The latter figure is probably more reliable because of the inevitable loss of water on the diaper, which is reflected in the relatively wide range of per cent of water.

Daily Fecal Excretion of Normal Infants and Children.—The observations recorded in table 5 were made primarily for the purpose of establishing standards of comparison with patients presenting the celiac syndrome. The analyses of fat were made by the Sperry method, using 3

LE 5.—*Daily Fecal Excretion of Normal Infants: Wet and Dry Weight of Feces, Total Fat, Neutral Fat and Fatty Acids (Sperry Method)*

	2 - 6 mo.		6 - 12 mo.		1 - 2 yr.		2 - 6 yr.	
ber of patients	11		10		10		11	
	Mean ± S. D.	Range	Mean ± S. D.	Range	Mean ± S. D.	Range	Mean ± S. D.	Range
weight, Gm./day	17.2 ± 8.8	6.6 - 37.0	25.7 ± 10.5	8.1 - 45.1	34.6 ± 14.7	16.7 - 54.1	25.7 ± 15.5	6.6 - 48.5
d weight, Gm./day	4.1 ± 1.9	2.0 - 5.7	6.2 ± 2.4	2.1 - 9.8	7.5 ± 3.1	4.2 - 11.0	6.6 ± 3.6	2.0 - 12.9
ent water	74.3 ± 3.9	65.5 - 79.5	75.4 ± 3.5	67.5 - 79.8	77.5 ± 4.0	69.5 - 85.7	74.2 ± 7.2	62.3 - 83.8
l fat, Gm./day	0.80 ± 0.31	0.29 - 1.30	1.04 ± 0.41	0.54 - 1.61	0.95 ± 0.33	0.33 - 1.40	1.01 ± 0.51	0.35 - 1.79
per cent dried weight	22.1 ± 10.5	5.2 - 43.1	17.6 ± 4.9	11.0 - 25.5	13.7 ± 5.6	6.1 - 25.8	14.5 ± 2.7	9.4 - 18.9
ral fat, Gm./day	0.17 ± 0.09	0.11 - 0.38	0.291 ± 0.21	0.12 - 0.83	0.39 ± 0.14	0.15 - 0.59	0.37 ± 0.23	0.06 - 0.80
y acids, Gm./day	0.63 ± 0.26	0.14 - 0.97	0.75 ± 0.31	0.39 - 1.38	0.56 ± 0.25	0.18 - 0.84	0.55 ± 0.26	0.27 - 0.99
ral fat, % of total fat	21.9 ± 11.4	7.8 - 51.9	26.9 ± 12.0	13.1 - 50.1	43.2 ± 13.5	22.9 - 63.9	38.2 ± 8.0	19.3 - 45.0

mal infants and children. There is surprisy little difference in the various age groups. range of variation is influenced not only by technical difficulties discussed previously and ndividual variations but by the error inherent ollecting feces over so short a period as three s. The last-mentioned factor introduces less iation than might be expected. Within a e range of normal foods the diet also intros relatively little variation when the infant ormal, although it often has a marked effect n the patient has digestive difficulty. Accordto my data, any figure which is more than e the mean of the normal is outside the range normal and hence represents a significant erence (table 5). The determination of total y fecal excretion of fat is therefore of real ortance in the clinical study of chronic indiion. Normally the fat also forms a fairly stant proportion of the solid material of the s if age is taken into consideration. In drops of acid and testing with litmus. These observations were made before the advisability of further acidification was appreciated. However, in comparing analyses made with 3 and with 10 drops of acid it was found that for feces of normal fat content the difference was slight. Both methods were used on 9 duplicate samples from patients with normal excretion of fat; the means of the difference between pairs was 0.20 Gm. of total fat per day; the mean figure for the total fat with 3 drops of acid was 1.37 Gm. and with 10 drops 1.56 Gm. This error, of about 16 per cent, is not sufficient to invalidate the use of the figures given in table 5 as a basis of comparison with the much larger figures for patients with steatorrhea.

The data in table 5 are also of interest in bringing out differences in fecal excretion due to age during the first few years of life. These changes are most marked in the two younger groups. For the age group of 2 to 6 months the total stool is smaller, with a relatively greater amount of fat and a smaller proportion of neutral fat than for the older age group. In all these

. Miller, R.; Webster, J., and Perkins, H.: Celiac ntilism: Its Fat Digestion and Treatment by Bile s, Lancet **2**:894-987, 1920.

respects my findings are intermediate between the values of Gordon and McNamara [12] for newborn infants and those obtained for the infants over 6 months of age in the present series. The amounts of feces and of total fat excreted daily are closely similar for the groups between 6 months and 6 years. It would be interesting to know whether the differences between the group under 6 months of age and the three older groups are due entirely to inclusion of solids in the diets of the older infants or are due in part to changes resulting from increased age.

Evaluation of the Reliability of Microscopic Examination of Feces for Fat.—The chemical methods which have been discussed are too expensive, time consuming and slow for routine clinical use. Microscopic examination of feces gives an immediate answer with a few minutes' labor. If reliable, even though only so far as to give no false negative results, this method could be used to screen out the patients who do not have steatorrhea and hence to leave a small number, for whom examination gives positive or equivocal results, for more careful study by chemical methods. In order to test the reliability of the microscopic estimate of fecal fat, a comparison was made of microscopic estimates and the results of chemical determinations based on 129 three day collections of stools from subjects with various clinical syndromes and of various ages, including infants, children and a few adults.

The samples were rated 0 to ++++ by the criteria for microscopic examination previously described. When any method involving estimation in this way is used, the human element enters in to a large degree. The present observations were made by four persons, all without much previous experience in the method. Each observer was checked on the first half-dozen examinations and on occasional later doubtful ones, but most of the data were based on one observer's opinion. The error so introduced was increased by a tendency of one of the observers to fear false positives and of another to fear false negatives. It is probable that the results would have been somewhat more consistent if all examinations had been made by the same person.

Two precautions were found advisable early in the experiment. One of these was the necessity of avoiding the presence of particulate matter, especially granules of dye, in the sudan IV solution. This was done by filtration of fresh solutions, by avoiding agitation of the dropping bottle and by observing the clarity of the solution from time to time. Six observations [illegible] discarded when it was discovered that use[illegible] new batch of unfiltered solution uniformly g[illegible] ++++ grading to all specimens examine[illegible]

The second precaution was based on re[illegible] tion that certain samples of feces with e[illegible] fat do not have many droplets of fat but in[illegible] show crystals or flakes; moreover, these c[illegible] tals or flakes of fat may stain only faintly [illegible] sudan IV and are sometimes dissolved in[illegible] alcoholic solvent of the stain. For this re[illegible] the custom was adopted of mixing the st[illegible] material with a drop or two of an aqueous [illegible] tion. If crystals or flakes were present [illegible] solubility in dehydrated alcohol U. S. P. [illegible] readily tested under the microscope.

The estimates made by microscopic exam[illegible] tion were compared with the per cent of [illegible] weight as total fat (as determined by the Sp[illegible] method), the number of grams of fat excreted [illegible] day and the per cent of fat as neutral fat. The [illegible] of these comparisons showed good correla[illegible] the second fair and the third poor (table[illegible] and 7). It is safe to say that if the samp[illegible]

TABLE 6.—*Frequency Table Showing Correlation tween Microscopic Estimate of Fat and Total F as Per Cent of Dried Weight of Feces as Determined by Sperry Method* *

Total Fat, Per Cent of Dried Weight	Microscopic Estimate				
	0	+	++	+++	++
<5	0	0	0	0	
5-10	8	0	2	0	
10-15	18	2	3	0	
15-20	21	2	3	0	
20-25	11	2	4	1	
25-30	8	1	0	6	
30-35	0	1	3	5	
35-40	0	1	0	1	
40-45	0	1	1	1	
45-50	0	0	1	3	
50+	0	0	0	1	

* The upper limit of normal is 25 per cent.

TABLE 7.—*Frequency Table Showing Correlation tween Microscopic Estimate of Fat and Total Excretion of Fat per Day, in Grams* *

Total Fat, Gm./Day	Microscopic Estimate				
	0	+	++	+++	++
0-2	44	3	6	4	
2-4	16	4	7	5	
4-6	4	2	0	4	
6-8	2	0	3	3	
8-10	0	0	1	1	
10+	0	1	0	2	

* The upper limit of normal is 2 Gm. per day for [illegible] under six years.

graded 0 or + by microscopic examination total fat present does not exceed 30 per cent the dried stool. If the sample is graded ++ or ++++, there is an excess of fat in stool; that is, the fat comprises more than

12. Gordon, H. H., and McNamara, H.: Fat Excretion of Premature Infants: Effect on Fecal Fat of Decreasing Fat Intake, Am. J. Dis. Child. **62**:328-345 (Aug.) 1941.

ent of the dried weight, which is the upper of normal (table 6). If it is graded + or the examination should be repeated on other les. The instances in which a 0 grading iven a stool having over 25 per cent of fat +++ to ++++ grading was given a le containing less than 25 per cent of fat then studied. For the 8 patients whose les were graded 0 the daily excretion of fat s follows: 1 patient, 7.14 Gm.; 5 patients, een 2 and 4 Gm., and 1 patient, less than n. The first of these patients is the only or whom the results on microscopic exami- n might have been clinically misleading. the 3 patients for whom the samples graded +++ or ++++ and contained than 25 per cent of dried feces as fat 1 ed a daily excretion of less than 2 Gm.; figures for the other 2 patients were 11.8 12.5 Gm. respectively. Again, there was 1 patient for whom the grading might have o error in diagnosis. The correlation be- n microscopic fat and daily fecal excretion at is somewhat less close than might be cted. Some patients with pancreatic de- ncy who were eating poorly of a diet con- ng fat, usually protein milk, produced scanty of high relative fat content but a low total excretion of fat; patients with the celiac rome often produced voluminous stools of al relative fat content but with high absolute es for daily excretion of fat. Correlations per cent of fat as neutral fat are poor, since al persons, especially infants, may excrete all amount of fat, most of it as unsaponifiable rial in the neutral fat fraction, and the micro- ic examination of these samples is negative fat; on the other hand, patients who excrete excess of total fatty acids may have a low tive figure for neutral fat and a microscopic ling of +++ or ++++. The samples all the patients with pancreatic deficiency ived gradings of +++ or ++++ when were receiving a normal diet. One can only clude that the formations identified in stained arations as "neutral fat droplets" may or not be composed of neutral fat, since they be present when the neutral fat is within normal range.

n the clinical use of microscopic examination he stool for fat the actual grading of the speci- n is of little value and may be misleading un- the dietary intake of fat, the possibility of dication with mineral oil and the general con- on of the patient are considered. No patient been encountered for whom a diet low in fat h skim milk resulted in stools graded +++ ++++ for fat; this observation includes the patients with pancreatic steatorrhea and those with celiac disease associated with marked inability to utilize fat.

If the technical sources of error are avoided, the microscopic examination for fat of stools obtained from patients with a normal intake of fat gives an estimate of fat excretion which has a high degree of reliability. In the present observations this method would have led to an erroneous diagnosis for 2 of 85 samples graded 0 or +++ to ++++. Equivocal readings of + or ++ permit no prediction.

SUMMARY AND CONCLUSIONS

In a comparison of the Fowweather and Sperry methods for the determination of fecal lipids, with an analysis of the sources of error of both methods, the Sperry method has been found more reliable.

The daily fecal excretion of a series of normal infants and children receiving normal diets has been studied. Mean figures are given for wet weight of stools, dry weight, total fat content and neutral fat and fatty acid fractions.

Determination of total excretion of fat per day is of clinical importance in the study of chronic indigestion and can be made with a fair degree of accuracy.

Changes in lipid partition have been found to occur in samples of stools as ordinarily collected; these changes reflect neither analytic error nor metabolic activity but may lead to erroneous conclusions in regard to the patient's digestive efficiency.

Neutral fat is hydrolyzed in vitro while feces are standing at temperatures as low as 6 to 7 C. For this reason analysis of feces of patients with pancreatic steatorrhea may show no relative excess of neutral fat and hence may be misleading. The proportion of neutral fat, including the unsaponifiable fraction, may be high for patients with good absorption of fat and low for those with pancreatic steatorrhea. The converse is true of the figure for total fatty acids.

Determination of soap by the Fowweather method is unreliable. The amount of fatty acids combined to form soap reflects the p_H of the feces, which can be more accurately determined by other means.

Comparison of the estimate of the amount of fecal fat by microscopic examination with the results of chemical examination in 129 instances reveals a high degree of correlation when the microscopic gradings were 0 or +++ to ++++. Intermediate gradings cannot be interpreted accurately. The method is useful for screening out patients without steatorrhea.

The Babies Hospital.

PLACE OF CONTACT AND RADIAL SPREAD OF EPIDEMIC POLIOMYELITIS

ALBERT E. CASEY, M.D.

BIRMINGHAM, ALA.

The most severe epidemic of infantile paralysis on record in the state of Alabama occurred during the summer and autumn of 1941, the greatest concentration of cases being in rural Walker County. One hundred and twenty-one cases in Walker County were officially reported during the epidemic; 4 sporadic cases which occurred after the epidemic had subsided were also reported. Special studies on the epidemiology and other phases of the disease were made in the county at that time.

MATERIALS OF THE STUDY

It was found during the study that the incubation period from the day of exposure until the onset of the prodromal period averaged twelve days.[1] The infectious period was found to be between three days before and three days after the onset of the prodromal period.[2] Of 101 cases of poliomyelitis in which the families of the victims were satisfactorily interviewed, approximately 80 per cent could be traced to a contact averaging several hours in duration with a person or persons in whom the disease was in the infectious period.[2] There was evidence that the prolonged contacts during which transmission of the disease apparently occurred were not all place contacts: Many person to person contacts were scattered about the county. Most of the cases could be traced directly or indirectly to one focus at Barney; the present paper analyzes the relation of the various secondary places of contact to this original site.

The epidemic was considered to have begun on the day of exposure of 2 persons to an infected person on the day of the onset of her prodromal period, which was June 19-20. For purposes of analysis, the epidemic was divided into periods as follows: The days on which exposure of 2 persons to an infected person took place (June 19 and 20) were taken as the first period; from June 20 the remaining days of the epidemic were divided into six twelve day periods (twelve days being the average length of the incubation period) and one twenty-four day period—the last period being extended in order to reduce the sampling error. The epi was considered to have ended on September 23.

Of the 121 cases officially reported during the demic, 2 were excluded from consideration be after more critical study the diagnoses were consi doubtful. However, the total number of cases st was kept at 121 by the addition of 2 officially unre but unquestionable cases with single exposures, have been mentioned in a previous publication.[1] E sive of the original case, the series was divided into groups—those in which a single exposure occurred cases), those in which there were multiple expo (45 cases) and those in which the patient's history not considered adequate or in which there was no hi of direct contact (38 cases).

OBSERVATIONS

Places of Contact in Cases in Which There Known Exposure to Infection.—Places of contact be established in 37 instances in each of which was a history of a single exposure to an infected son during the infectious period. These sites of e sure, along with the location of the original case, plotted on a map, a code being used to indicate in w period the contact occurred (fig. *A*). In 45 inst in which there were multiple exposures to 1 or infected persons during the infectious period, place contact could be determined. In 2 cases patients so posed had had more than one site of exposure; h the places of contact were not plotted, as were t in the remaining 43 cases (fig. *B*).

In 68 of the 80 cases in which the places w single or multiple exposures took place were pl the infection could be traced through apparent cessive generations of direct contact to the original In the other 12 cases the patients had been exp to infected persons for whom no history of direct indirect association with the Barney group could obtained. The air line distance between the place contact in each of the 68 cases and the original si infection at Barney was determined. The average tance for the places of contact corresponding to period (see figs. *A* and *B*) were as follows: 1, 1.7, 4.3, 6 and 7.6 miles respectively for periods 2, 3, 4, and 7 and 8.7 miles for period 8. When these ave distances were plotted, a series of expanding concen circles was obtained (fig. *C*—solid line circles). It be noted that the average distance of the place of posure to an infected person from the original site each period moved radially from the original site at rate of about 1 mile (1,600 meters) to each ten or 8.7 miles (about 14,000 meters) during the nin six days of the epidemic.

The entire 80 cases in which there was a his of single or multiple exposure, including those w could not be traced to the site of infection at Bar together with the original case at Barney, were analyzed as to sites of exposure, as is shown in accompanying table.

From the Department of Pathology, Baptist and Jefferson Hospitals, Birmingham, Ala., and the Louisiana State University School of Medicine, New Orleans.

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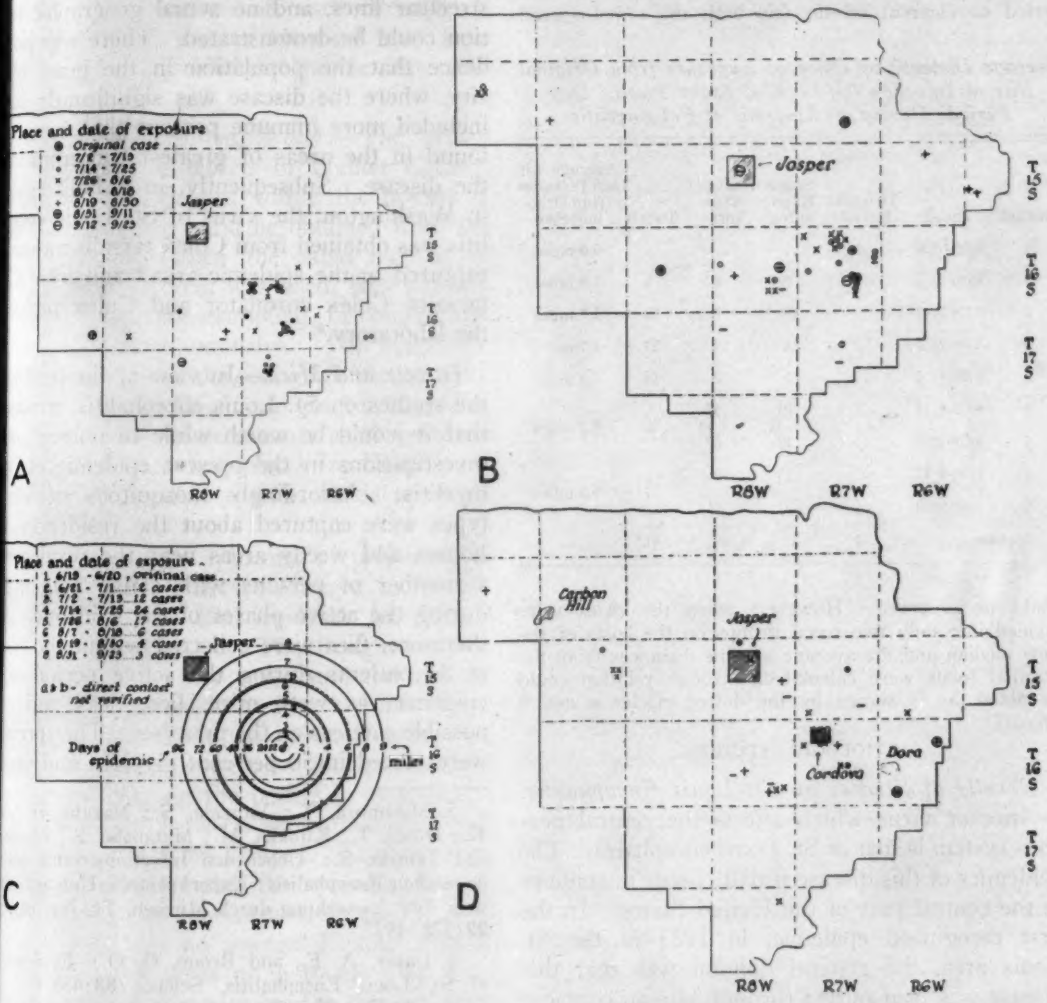
1. Casey, A. E.: The Incubation Period in Epidemic Poliomyelitis, J. A. M. A. **120**:805-807 (Nov. 14) 1942.

2. Casey, A. E.: Observations on an Epidemic of Poliomyelitis, Science **95**:359-360, 1942.

bable Places of Contact in Cases in Which There No Known Exposure to Infection.—In 38 cases tory of direct contact with an infected person or is was obtained; of these, 12 were not studied in because of lack of time; the patients in 3 cases residing outside the county in another epidemic (1 in Birmingham, Ala., and 1 in Tennessee) the disease was probably being contracted; in es patients 16 years of age or older (the only ts in the entire series in this age group) had ngly had no exposure to infected persons, but 3 em had traveled widely within and 3 had traveled n and without the county, and a satisfactory history which it is assumed that the disease may have been transmitted. Two others went swimming in a stagnant stream beside which were located the residences of a number of patients. In 1 patient, an infant who lived 7 miles from the original focus, poliomyelitis developed within the usual incubation period after her father, who was a painter, had been working in the homes of several other patients at Barney during the infectious period of the disease. It was the father's custom to hold the infant in his arms immediately on arriving home, before he had changed clothes. One child, about 2 years old, stayed daily in a small store near Jasper and came in constant contact with customers. Five others gave his-



A, places of contact in 37 cases in which a single exposure to an infected patient took place, with the site of the inal case at Barney. Symbols indicate in which period the contact occurred. *B*, sites of exposure in 43 cases which there was history of multiple contact. *C*, radial spread of poliomyelitis. The circles indicate average line distances from the original focus at Barney. *D*, location of the patients twelve days prior to the onset he prodromal period in 32 cases in which there was no history of contact or in which the history was not ained.

The following symbols are used: cross in circle, period 1; dot in circle, period 2; black circle, period 3; white le, period 4; ×, period 5; —, period 6; +, period 7; +, in circle, period 8a (twelve days); —, in circle, iod 8b (twelve days); T, township; R, range; S, south, and W, west. Each township is 6 miles square.

d not be obtained. The patients in the remaining cases were carefully studied, and no history of direct tact with infected persons during the infectious iod could be obtained. Eight of the 17 lived within w hundred yards of infected persons or had visited he neighborhood of such persons during the period in tories of human contacts, but none reported contacts with persons who had febrile illnesses or frank poliomyelitis during the three weeks prior to the onset.

The 3 cases in which the disease had possibly been contracted while the subject was outside the county and the 3 in which persons 16 years of age had traveled

outside as well as within the county were omitted from consideration, and the remaining 32 cases, which had been inadequately studied or in which no history of contact could be obtained, were analyzed as to possible correlation with radial spread. Since the average incubation period had been found to be twelve days, it was assumed for statistical purposes that the probable site of exposure was the location of the patient twelve days prior to the onset of the prodromal period. This location was determined for each of the 32 cases and the probable sites of exposure plotted, the same codes being used for each period as before (fig. *D*). When a calculation was made of the average air line distances from the original focus for the cases commencing in each period no clearcut relationship with the radial spread could be observed. However, when the cases were divided into only two equal groups on the basis of the time median and the average air line distances from the original focus were calculated, a rough relation could be noted, as is shown by the dotted circles *a* and *b* (fig. *C*).

Average Distance of Place of Exposure from Original Site of Infection for Each of Eight Twelve Day Periods During an Epidemic of Poliomyelitis

Period	Dates	Original Patient	Single Exposures	Multiple Exposures	Totals	Average Air Line Distance from Original Site
1	6/19-6/20	1	..	..	1	0.0 miles
2	6/20-7/ 1	..	0	2	2	1.0 miles
3	7/ 2-7/13	..	10	2	12	1.7 miles
4	7/14-7/25	..	14	13	27	4.7 miles
5	7/26-8/ 6	..	7	12	19	7.3 miles
6	8/ 7-8/18	..	2	4	13	8.2 miles
7	8/19-8/30	..	2	5		
8[a]	8/31-9/11	..	1	3	7	9.9 miles
8[b]	9/12-9/23	..	1	2		
Totals.....		1	37	43	81	

ETIOLOGIC STUDIES

Results of Studies on St. Louis Encephalitis.—Another virus which affects the central nervous system is that of St. Louis encephalitis. The epidemics of this disease usually occur in summer in the central part of the United States. In the first recognized epidemic, in 1933 in the St. Louis area, the general opinion was that this disease was transmitted through human contact,[3] but one worker suspected the mosquito.[4] No definitive statistical studies were reported. Later, transmission of St. Louis encephalitis by Culex pipiens was reported after a study in which laboratory methods were used.[5] W the disease recurred in 1937, an epidemiol study of both the 1933 and the 1937 outb was conducted[6] with use of modern method biometric analysis.[7] The foci of the disease found not to radiate but to remain stationar areas containing weedy and sewage-cont nated streams, like the areas which formed breeding grounds for Culex mosquitoes. correlation with the routes of human travel, cluding highways, boulevards and railway streetcar lines, and no actual geographic ra tion could be demonstrated. There was no dence that the population in the heart of city, where the disease was significantly sca included more immune persons than were to found in the areas of greatest concentration the disease. Subsequently, in a 1941 epide in Washington, the virus of St. Louis encep litis was obtained from Culex tarsalis mosqui captured in the epidemic area.[8] and from C tarsalis, Culex coronator and Culex pipiens the laboratory.[9]

Insects and Mice.—In view of the findings the studies on St. Louis encephalitis, it was that it would be worth while to pursue sim investigations in the present epidemic of po myelitis. Accordingly mosquitoes of vari types were captured about the residences, houses and weedy areas near the residences a number of persons with active poliomyel during the active phases of the epidemic. F thermore, flies were collected within the hou of 34 patients during the active period of epidemic, as were mice, lice, fleas and ot possible carriers on the premises. The specim were placed in 50 per cent glycerin and isoto

3. Leake, J. P., and others: Report of the St. Louis Outbreak of Encephalitis, Bulletin 214, United States Treasury Department, Public Health Service, 1935. McCordock, H. A.: The St. Louis Epidemics of Acute Encephalitis, Washington Univ. M. Alumni Quart. **1**: 141-147, 1938. Smith, J. E.: Discussion of St. Louis Encephalitis, South. M. J. **35**:79-80, 1942.

4. Lumsden, L. L.: Epidemiology of St. Louis Encephalitis, United States Public Health Service, 1933, unpublished report.

5. Mitamura, T.; Yamada, S.; Nazato, H.; M K.; Hosoi, T.; Kitoaka, M.; Matanabe, S.; Okubo, and Temjin, S.: Ueber den Infectionsmodus der demischen Enzephalitis: Experimentelle Untersuchun über ihre Ansteckung durch Mücken, Tr. Jap. path. **27**:573, 1937.

6. Casey, A. E., and Broun, G. O.: Epidemiol of St. Louis Encephalitis, Science **88**:450-451, 1 Casey, A. E.: The Geographic Pathology of St. L Encephalitis, South. M. J. **35**:869-880, 1942.

7. Fisher, R. A.: Statistical Methods for Resea Workers, ed. 4, Edinburgh, Oliver & Boyd, Ltd., 1

8. Hammon, W. M.; Reeves, W. C.; Brookman, Izumi, E. M., and Gjullin, C. M.: Isolation of Viruses of Western Equine and St. Louis Encephal from Culex Tarsalis Mosquitoes, Science **94**:328-3 1941; Mosquitoes and Encephalitis in the Yaki Valley, Washington, J. Infect. Dis. **70**:263-266, 19

9. Hammon, W. M., and Reeves, W. C.: Cu Tarsalis Coq. a Proven Vector of St. Louis Encep litis, Proc. Soc. Exper. Biol. & Med. **51**:142-143, 19 Reeves, W. C.; Hammon, W. M., and Izumi, E. Experimental Transmission of St. Louis Encephal Virus by Culex Pipiens Linnaeus, ibid. **50**:125-128, 19

ion of sodium chloride and kept at 30 F. they were pooled and extracts inoculated mulatta monkeys. No evidence of the dis- developed in the monkeys.[10] No success entifying a possible insect vector was ob- d in this 1941 epidemic in Walker County for the finding of the virus in a batch of flies ed near a privy used by the families of 4 nts with acute poliomyelitis.[11] The latter ng was probably not significant, for the of poliomyelitis could not be demonstrated , with the same technic used throughout, were trapped in this identical spot after disease had subsided in the families who the privy.

agnant and Polluted Water.—The failure nd the virus in insects in Walker County, ially in flies trapped within the houses of nts, although it could be obtained from the s of patients when the same method of rvation in glycerin was used, was possibly ficant, since the sampling of mosquitoes flies was fairly extensive. The possibility rred to mind that the sewage or the stagnant ms in Walker County might be related to myelitis in the same manner as the streams h furnished breeding places for Culex mos- es were related to the St. Louis epidemic ncephalitis.

early every large epidemic of poliomyelitis occurred after a severe drought in the af- d area, and the 1941 epidemic in Walker nty was no exception. The drought was the t in more than fifty years, and the level of Warrior River was at its lowest point. Since e was a dam below the epidemic area, this r and its tributaries were not much more than nant pools during the period in question.

he connection between water and epidemic myelitis cannot be escaped. Studies done Louisiana revealed that the epidemic disease three times as concentrated in towns with ater supply but no sewage disposal system t was in rural areas or in towns with both r supply and adequate sewage disposal sys- s.[12] Detailed studies [13] in Walker County in l showed the same condition. The epidemic was much more severe in Barney and Cordova and such primitive communities than it was in Jasper, which has both a municipal water supply and a sewage disposal system. The same contrast was observable in communities outside Walker County where the epidemic raged. The finding of the virus of poliomyelitis in sewage [14] and streams polluted by sewage suggests factors for the inception of an epidemic.

In Louisiana no incrimination of the supply of drinking water could be found. In Walker County swimming or other contacts with polluted water could have been a factor in some cases. Significantly, most of the older children or young adults who had not given any history of contact with an infected person had nevertheless been bathing in one of the sewage-polluted streams. Also, the earliest cases of poliomyelitis in Alabama occurred in the county north of Walker County (Cullman) in February, March and April of 1941. The drainage from this portion of Cullman County is into the stagnant Warrior River, on the bank of which the child who gave rise to the epidemic resided. This child had been bathing with her parents in the river on several occasions prior to her illness. With these exceptions, however, the great majority of the infants had not been in contact with sewage-polluted streams or other water. In fact, almost none of the patients for whom a history of contact was obtained had had such an experience, inasmuch as the ages of the infants were largely between 6 months and 4 years.

Feces.—The proved presence of the virus of poliomyelitis in the stools of hospital patients and the almost complete absence of secondary cases from such institutions cast grave doubt on the role of feces in the transmission of the disease after paralysis has occurred. This opinion was confirmed in Walker County by the almost complete absence of infection following exposure to infected persons after the early prodromal period.

Human Carriers Not Affected with the Disease.—The Walker County studies also cast doubt on the role of adults or of persons not clinically ill in transmitting the disease.[15]

Protracted Human Contacts.—The type of contact which took place in the cases of poliomy-

. Casey, A. E., and Wenner, H. A.: Unpublished

. Paul, J. R.; Trask, J. D.; Bishop, M. B.; Mel- J. L., and Casey, A. E.: The Detection of Polio- itis Virus in Flies, Science **94**:395-396, 1941.

. Casey, A. E., and Aymond, B. J.: The Distri- on of Poliomyelitis in Louisiana, Science **91**:17, ; The Geographic Distribution of Disease: III. Decade of Poliomyelitis in Louisiana, Pub. Health **55**:1295-1300, 1940.

. Casey, A. E., and Aymond, B. J.: Unpublished ies.

14. Paul, J. R.; Trask, J. D., and Culotta, C. S.: Poliomyelitic Virus in Sewage, Science **90**:258-259, 1939. Kling, C., quoted by Levaditi, C.: Sur la présence du virus poliomyélitique dans les eaux d'égout, Bull. Acad. de méd., Paris **123**:335-337, 1940. Paul, J. R., and Trask, J. D.: The Virus of Poliomyelitis in Stools and Sewage, J. A. M. A. **116**:493-498 (Feb. 8) 1941.

15. Wenner, H. A., and Casey, A. E.: A Community Study of Carriers in Epidemic Poliomyelitis, J. Clin. Investigation **22**:117-125, 1943. Casey.[2]

elitis studied was not casual, as is often true with measles or mumps. The children in the Walker County group averaged several hours of play together. Such a long average period of contact seems to indicate that droplet infection would be a difficult explanation to rely on in the usual case. The long period of contact makes it seem more logical to incriminate hands contaminated with secretions of the nose or mouth or with excreta, exchanged in play among small children. Studies have shown that the virus of poliomyelitis is present in the nose, mouth and throat during the first day or two of the disease but not subsequently.[16] The suggestion of Colmer that teething may be a factor[17] recalls the drooling of infants of that age. Furthermore, the contact seems as likely to have come about from the infected child's visiting the residence of the victim as from the reverse.[2]

SUMMARY AND CONCLUSIONS

In a rural epidemic of poliomyelitis, minutely studied, there was evidence of patient to patient contact in 80 per cent of the cases in which an adequate history was obtained. When the average air line distances of the places of contact for each twelve day period of the epidemic were calculated, there was evidence of orderly radial spread at the rate of about 1 mile (1,600 meters) for each ten or eleven days, or an average of 8.7 miles (about 14,000 meters) in the ninety-six days. The contacts were almost entirely among children under 15 years of age and very largely among children under 4 years of age. No histor contact were obtained for older children adults. The probable site of exposure of tients whose history was inadequate or wa obtained was defined as the location of th tient twelve days prior to the onset of the dromal period; the average air line distan these sites from the original focus fitted ro into the lines of spread.

Contacts with healthy persons and with drinking water, flies, mosquitoes, roden other vectors did not seem to have much i tance in the Walker County epidemic. Thi further substantiated by the radial s through patient to patient contacts in the W County epidemic of poliomyelitis, which trasted to the apparent absence of such spread in two epidemics of St. Louis encepl a neurotropic summer virus, carried by mosquitoes. There was the possibility sewage-polluted waters concentrated by a drought explained many sporadic cases, cases of the disease in adults and, perhap original case from which the epidemic st Is it possible that the summer incidence o demic poliomyelitis depends usually on the centration of sewage-polluted streams and w by drought and heat in areas where ther sufficiently large infant populations and num prolonged contacts among infants? Su might thus initiate, and flies and adult co sporadically facilitate, epidemics, which are moted largely by prolonged contacts children in the late incubation and early dromal stages of poliomyelitis. Oral, nasa pharyngeal secretions and feces-contami hands seem to be highly infectious in thes not in later stages of the disease.

Baptist and Jefferson Hospitals.

16. Paul, J. R.; Trask, J. D., and Webster, L. T.: Isolation of Poliomyelitis Virus from Nasopharynx, J. Exper. Med. **62**:245-257, 1935. Howe, H. A.; Wenner, H. A.; Bodian, D., and Maxcy, K. F.: Poliomyelitis Virus in the Human Oropharynx, Proc. Soc. Exper. Biol. & Med. **56**:171-172, 1944.

17. Colmer, G.: Paralysis in Teething Children, Am. J. M. Sc. **5**:248, 1843.

ASCORBIC ACID, RIBOFLAVIN AND THIAMINE CONTENT OF CHOCOLATE MILK

ARTHUR D. HOLMES, PH.D.; CARLETON P. JONES, M.S.; ANNE W. WERTZ, B.S., AND W. S. MUELLER, PH.D.

AMHERST, MASS.

is difficult to estimate the volume of choco- milk consumed annually in the United States, nationwide survey [1] of the industry showed stores, homes and schools are the principal s, that middle westerners consume the larg- mounts and that 80 per cent of the choco- milk is consumed by persons who do not plain milk. Thus chocolate milk does not ce plain milk but is consumed by persons otherwise would not obtain the nutrition ied by milk. A house to house canvass of average American homes revealed that 80 ent of the mothers and 90 per cent of the rs never drank milk and that after 12 years e 60 per cent of the children discontinued ing milk. It is from such homes that a large ortion of the consumers of chocolate milk , and they are attracted by the popular olate flavor.

s a matter of fact, chocolate milk is usually with cocoa and contains no chocolate. equently, if such a product were labeled olate milk it would be legally an adulterated misbranded product. However, the term olate milk is commonly used for the product ared with cocoa, and it will be so used in discussion.

hile a number of investigations have sup- information concerning some phases of the itive value of chocolate milk, a review of literature produced no experimental data erning the value of chocolate milk as a source tamins. Accordingly, it was decided to deter- e the amounts of ascorbic acid, riboflavin and mine in freshly made chocolate milk.

METHODS AND MATERIALS

e chocolate milk used in this study was prepared ixing cocoa syrup with pasteurized whole milk essed as described by Holmes.[2] The composition of the cocoa syrup was 45.2 per cent water, 8.2 per cent cocoa powder, 46.2 per cent sugar and 0.4 per cent salt. Thirty cubic centimeters of the cocoa syrup was thoroughly mixed with 240 cc. of pasteurized whole milk that had been produced during the preceding eighteen hours. While chocolate milk may be prepared with condensed or evaporated milk or with buttermilk and is frequently made with skimmed milk, only whole milk was used for the purpose of this study.

Two types of cocoa were used, American process and Dutch process cocoa. It will be noted from the composition of these two cocoas, reported in table 1, that the greatest difference is in the amount of tannic substances which they contain. Mueller [3] in his study of the significance of tannic substances and of theobromine in chocolate milk found that a cocoa powder which contained 12.15 per cent of tannic substances was more toxic to rats than one that contained 2.67 per cent when the two powders had the same amount of theobromine and caffeine.

TABLE 1.—*Results of Analyses of Cocoa Powders*

Constituents	American Process, per Cent	Dutch Process, per Cent
Moisture	5.40	4.32
Protein *	22.62	20.31
Fat	11.23	10.32
Fiber	5.25	5.33
Other N-free matter	35.50	43.11
Ash	5.98	11.60
Theobromine + caffeine	1.87	2.34
Tannic substances	12.15	2.67

* Nitrogen (minus theobromine and caffeine nitrogen) × 6.25.

The process of manufacture is in general the same for the two types of cocoa. The dried beans of the tropical tree Theobroma cacao are cleaned and carefully roasted. To make chocolate, which contains approximately 50 per cent of fat, the beans are ground to a paste and run into molds. To make cocoa by the American process a considerable portion of the fat is removed by subjecting the ground beans to hydraulic pressure between heated plates and grinding the residue to a fine powder. In the Dutch process the finely powdered cocoa is treated with alkali in an attempt to render the cocoa more "soluble" or more "digestible." The treatment with alkali reduces the content of fat somewhat, increases the content of theobromine, greatly increases the content of ash and produces a much darker cocoa.

The hydrogen ion concentrations of the cocoa syrups prepared from the two types of cocoa used in this study were quite different, p_H 5.05 for the cocoa syrup made

ontribution 531, Massachusetts Agricultural Experi- Station.

. Nationwide Survey Reveals Trends in Sale of olate Milk: What Are the Sales' Possibilities hocolate Milk? Milk Dealer **24**:38-41, 1935.

. Holmes, A. D.: Effect of Pasteurization on the flavin Content of Milk, J. Am. Dietet. A. **20**:226-1944.

3. Mueller, W. S.: The Significance of the Tannic Substances and Theobromine in Chocolate Milk, J. Dairy Sc. **25**:221-230, 1942.

with American process cocoa and p_H 7.12 for that made with Dutch process cocoa. However, this difference was not noticeable in the chocolate milk. The values were p_H 6.5 for chocolate milk made with American process cocoa and p_H 6.7 for chocolate milk made with Dutch process cocoa, as compared with p_H 6.6 for plain milk.

In order to eliminate as far as possible any effect of storage, the assays were started as soon as convenient after the mixing—the assay for ascorbic acid within fifteen minutes, that for riboflavin within an hour and that for thiamine within two hours. The amount of ascorbic acid was determined by a modification of the method reported by Holmes, Tripp, Woelffer and Satterfield.[4] Fifty cubic centimeters of chocolate milk was mixed with 30 cc. of acid (equal parts of 5 per cent metaphosphoric acid and 10 per cent of acetic acid) and diluted to 100 cc. A 60 cc. aliquot was centrifuged at high speed for ten minutes; then 0.5 Gm. of 300 mesh bentonite was added to the decanted solution, which was shaken vigorously for one minute and then filtered. Ten cubic centimeter aliquots of the filtrate were titrated with a standard 2,6-dichlorophenolindophenol dye solution. The riboflavin was determined by the procedure described by Holmes, Jones, Wertz and Kuzmeski,[5] except that potassium permanganate followed by solution of hydrogen peroxide was used instead of stannous chloride and sodium thiosulfate for discharging the chocolate color. The Hennessy [6] method was employed for determining the thiamine content of the chocolate milk.

RESULTS

The results of assays for the ascorbic acid, riboflavin and thiamine content of chocolate milk prepared with American process and with Dutch process cocoa are reported in table 2. The variations in the amounts of the vitamins under consideration were as follows: Ascorbic acid in the American process chocolate milk from 11.1 to 17.9 mg. and in the Dutch process chocolate milk from 5.2 to 14.2 mg. per liter; riboflavin in the American process chocolate milk from 1.38 to 1.71 mg. and in the Dutch process chocolate milk from 1.23 to 1.55 mg. per liter, and thiamine in the American process chocolate milk from 0.25 to 0.34 mg. and in the Dutch process chocolate milk from 0.21 to 0.28 mg. per liter. The average values obtained for the 16 samples were as follows: Ascorbic acid in the American process chocolate milk 15.4 mg. and in the Dutch process chocolate milk 11.5 mg. per liter; riboflavin in the American process chocolate milk 1.50 mg. and in the Dutch process chocolate milk 1.37 mg. per liter and thiamine in American process chocolate milk 0.31 mg. in the Dutch process chocolate milk 0.25 Judging by these results, one may conclude freshly made chocolate milk is a good sour ascorbic acid, riboflavin and thiamine, but chocolate milk made with Dutch process co

TABLE 2.—*Ascorbic Acid, Riboflavin and Thiam Content of Chocolate Milk*

Sample No.	Ascorbic Acid		Riboflavin		Thiamin	
	American Process, Mg./L.	Dutch Process, Mg./L.	American Process, Mg./L.	Dutch Process, Mg./L.	American Process, Mg./L.	Dutch Pro Mg.
1	17.1	13.1	1.46	1.31	0.29	
2	16.3	11.5	1.38	1.34	0.30	
3	16.9	10.5	....	1.35	0.33	
4	17.6	13.3	1.65	1.40	0.30	
5	15.5	11.7	1.71	1.55	0.33	
6	17.9	14.2	1.42	1.34	0.34	
7	16.9	12.0	1.48	1.34	0.33	
8	16.4	11.8	1.39	1.34	0.30	
9	11.1	5.2	1.46	1.37	0.32	
10	15.5	13.0	1.41	1.35	0.35	
11	12.6	6.9	1.59	1.55	0.32	
12	13.5	10.3	1.43	1.32	0.30	
13	15.3	12.6	1.48	1.23	0.33	
14	13.8	12.2	1.53	1.34	0.33	
15	15.2	12.9	1.49	1.41	0.33	
16	14.8	12.4	1.55	1.37	0.30	
Average	15.4	11.5	1.50	1.37	0.31	

contains definitely less of these vitamins t chocolate milk made with American proc cocoa. Since the values reported here were tained for chocolate milk at the time it compounded, they may not apply to choco milk which is stored for various periods bet it is consumed.

COMMENT

A comparison of the nutritive value of chocolate milk under discussion with that of co

TABLE 3.—*Ascorbic Acid, Riboflavin and Thiamin Content of Chocolate Milk and of Whole Milk*

	Ascorbic Acid, Mg./L.	Ribo-flavin, Mg./L.	Thi mi Mg
Chocolate milk (American process).....	15.4	1.50	0.3
Chocolate milk (Dutch process).....	11.5	1.37	0.3
Whole milk pasteurized *.....	19.7	1.51	0.3
Whole milk pasteurized *.....	18.4	1.37	0.4
Whole milk pasteurized †.....	16.6	1.50	0.3

* Data from Holmes, A. D.; Jones, C. P.; Wertz, A. W.; Esselen, K., and McKey, B. V.: The Ratio of Ascorbic, Nicotinic and Pantothenic Acids, Riboflavin and Thiamine in Summer Milk, J. Dairy Sc. 27:849-855, 1944.

† Data from Holmes, A. D.; Lindquist, H. G.; Jones, C. and Wertz, A. W.: Effect of High Temperature, Short-Time Pasteurization on the Ascorbic Acid, Riboflavin and Thiamin Content of Milk, J. Dairy Sc. 28:29-33, 1945.

parable whole milk produced by the same he of cows at intervals preceding and following t production of the whole milk used to make t chocolate milk will be found in table 3. Inspe tion of the data will reveal that the averag riboflavin contents of the chocolate milk and t

4. Holmes, A. D.; Tripp, F.; Woelffer, E. A., and Satterfield, G. H.: A Study of Breed and Seasonal Variations in the Ascorbic Acid Content of Certified Milk from Guernseys and Holsteins, J. Nutrition **17**: 187-198, 1939.

5. Holmes, A. D.; Jones, C. P.; Wertz, A. W., and Kuzmeski, J. W.: The Ratio of Ascorbic Acid, Riboflavin and Thiamin in Raw and Pasteurized Milk, J. Nutrition **26**:337-345, 1943.

6. Hennessy, D. J.: A Standard Thiochrome Assay for the Determination of Thiamine in Cereal Products, Cereal Chemists' Bull. **2**:1-5, 1942.

le milk are similar but that the chocolate contained definitely less ascorbic acid and mine than did the whole milk. The vitamin ontent of the chocolate milk was not deter-ed, but the American Medical Association [7] pointed out that the vitamin A content of chocolate-flavored milks may range from a ligible amount to slightly less than that of le milk, depending chiefly on the amount utter fat present. The Association also states the majority of dairies use partially defatted for manufacturing chocolate milk. It should remembered that when skimmed milk is used making chocolate milk, it will contain slightly calcium, phosphorus and vitamin B_1 and nificantly less vitamin A than will whole milk. Consequently, families that prefer chocolate to plain whole milk would do well to buy colate milk known to be made with pasteurized le milk or to prepare it at home with a colate or cocoa syrup and pasteurized whole milk. Obviously, the food value of the commercial or the homemade chocolate milk will vary according to the composition of the syrup and the amount of it that is incorporated into the chocolate milk.

7. Accepted Foods and Their Nutritional Significance, Chicago, American Medical Association, 1939, 241.

SUMMARY

Sixteen samples of chocolate milk were prepared by mixing 30 cc. of cocoa syrup and 240 cc. of pasteurized, fresh whole milk. The American process chocolate milk contained 15.4 mg. of ascorbic acid, 1.50 mg. of riboflavin and 0.31 mg. of thiamine per liter, and the Dutch process chocolate milk contained 11.5 mg. of ascorbic acid, 1.37 mg. of riboflavin and 0.25 mg. of thiamine. From a practical standpoint these results indicate that chocolate milk made with American process cocoa is superior as a source of vitamins to chocolate milk made with Dutch process cocoa. Since these results were obtained from assays made as soon as the chocolate milk was compounded, they may not apply to chocolate milk that is stored for varying periods before it is consumed.

Massachusetts State College and Agricultural Experiment Station.

Case Reports

MALIGNANT HYPERTENSION; CURE FOLLOWING NEPHRECTOMY

FOLLOW-UP REPORT OF THE CASE OF A CHILD

ROGER L. J. KENNEDY, M.D.* AND NELSON W. BARKER, M.D.‡
ROCHESTER, MINN.
AND
CAPTAIN WALTMAN WALTERS, MC-V(S), U.S.N.R.

During the last decade, 1934 to 1944, much clinical interest and experimental effort have been centered on unilateral atrophic pyelonephritis and renal ischemia as causative factors in the production of hypertension. Shortly after the report of the experimental observations of Goldblatt and his associates [1] in 1934 on the production of persistent elevation of systolic blood pressure by means of renal ischemia, there appeared in the literature numerous accounts of the results of nephrectomy in cases of unilateral renal disease and hypertension. Immediate reduction in blood pressure occurred in a considerable number of cases. However, Barker and Walters [2] in 1940 pointed out that although the immediate results in a number of cases were good, the ultimate result of nephrectomy in some cases was not satisfactory with regard to permanent lowering of the blood pressure.

In 1941 we [3] reported the case of a girl, 7 years of age, who had had hypertension which was classified as malignant. Restoration of the blood pressure to a normal level followed removal of an atrophic pyelonephritic kidney and continued for three months after the operation. In the original report of this case reference was made to 2 other cases of hypertension in children in which nephrectomy produced relief. One of these was reported by Butler [4] and the other by Barney and Suby.[5] The blood pressure in bo cases had remained normal for twenty mont when they were reported. Later reports co cerning these cases have not been encountere but it is reasonable to assume that the reducti of the blood pressures was permanent. In ord to evaluate the permanent effect of operati procedure in cases such as these, this follow-u report is made five years after operation w carried out in our case.

REPORT OF CASE

A girl 7 years of age was brought to the Ma Clinic on Oct. 3, 1939, because of headaches, high blo pressure and "kidney trouble." At 2 years of age s had begun to have malaise and headaches, which h increased in severity and frequency. For three wee before her admission to the clinic the headaches h been almost constant. Urinary frequency occasional had been present. The systolic blood pressure was sa to have been 180 mm. of mercury during an attack measles ten months previously. Five weeks before h admission the right eye had turned inward and h remained in this position. For the last few days s had complained of blurring of vision.

Physical Examination.—Physical examination revealed some enlargement of the heart to the left, accentuatio of the second aortic sound and firmness of the peripher arteries. Examination of the ocular fundi revealed narrowing, grade 3 to 3+ (on the basis of a scale o 1 to 4, on which 1 indicates the least and 4 the greates change), of the retinal arterioles; sclerosis, grade 1 and angiospastic narrowing, grade 3. There were ex tensive retinitis of the malignant hypertensive type hyperemia with edema of the disks of 5 diopters, cotton wool patches and hemorrhagic extravasations. Right convergent strabismus and homonymous diplopia were noted. A roentgenogram of the chest revealed that th arch of the aorta was prominent and tortuous. The systolic blood pressure varied between 170 and 225 mm

* Section on Pediatrics, the Mayo Clinic.

‡ Division of Medicine, the Mayo Clinic.

1. Goldblatt, H.; Lynch, J.; Hanzal, R. F., and Summerville, W. W.: Studies on Hypertension: I. The Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia, J. Exper. Med. **59**:347 (March) 1934.

2. Barker, N. W., and Walters, W.: Hypertension and Chronic Atrophic Pyelonephritis: Results of Nephrectomy, J. A. M. A. **115**:912 (Sept. 14) 1940.

3. Kennedy, R. L. J.; Barker, N. W., and Walters, W.: Malignant Hypertension in a Child: Cure Following Nephrectomy, Am. J. Dis. Child. **61**:128 (Jan.) 1941.

4. Butler, A. M.: Chronic Pyelonephritis and Arterial Hypertension, J. Clin. Investigation **16**:889 (Nov.) 1937.

5. Barney, J. D., and Suby, H. I.: Unilateral Renal Disease with Arterial Hypertension: Report of a Case Apparently Cured Following Nephrectomy, New England J. Med. **220**:744 (May 4) 1939.

6. Wagener, H. P., and Keith, N. M.: Diffuse Arteriolar Disease with Hypertension and the Associated Retinal Lesions, Medicine **18**:317 (Sept.) 1939

ercury; the diastolic blood pressure varied between and 178 mm. An excretory urogram revealed that outline of the pelvis, the calices and the ureter on right side was practically normal. On the left side e was but little evidence of opaque medium. A ograde pyelogram revealed a small but dilated renal is on the left side with short, clubbed calices. This ure suggested chronic atrophic pyelonephritis.

peration on Oct. 13, 1939.—On Oct. 13, 1939, the kidney, which weighed 9 Gm., was removed. Micro-ic examination disclosed pyelonephritis, grade 3. glomeruli were fibrotic and hyalinized. The tubules e atrophic. The walls of the blood vessels were kened; in some vessels the lumen was obliterated. mediately after operation the blood pressure was 156 . of mercury systolic and 100 mm. diastolic; the next the reading was 134 mm. of mercury systolic and 100 . diastolic. The blood pressure dropped progressively il it reached a normal level, on about the tenth day. this time the changes in the retinas began to recede. child was permitted to return to normal activity.

eexamination Three Months After Operation.—ee months after the operation the patient was re-mined. She had been feeling well, and there had n no return of headaches. The strabismus had rected itself, and she had gained 8 pounds (3.6 Kg.). blood pressure at that time was as follows: left n, 108 mm. of mercury systolic and 74 mm. diastolic; ht arm, 102 mm. of mercury systolic and 70 mm. stolic. There was no rise in blood pressure during cold pressor test. Examination of the eyes re-led vision of 6/5 in the right and 6/6 in the left . The pupils, reflexes and visual fields were nor-. The retinal arterioles were generally narrowed, de 1 to 1+, and there was minimal sclerosis of onic type. There was no visible localized spasm or tspastic sclerosis. Examination revealed exudates idual to the previous edema, some of which were p and plaquelike, scattered in and about the macula the retina. There was slight increase of residual nective tissue on the optic disks. The disks were ; no active retinal lesions were seen. This examina-n indicated an excellent recovery.

Reexamination on May 31, 1940.—Another examina-n was made on May 31, 1940, about seven and a half nths after the nephrectomy, because the patient had slight cough. The chest was found to be clear. A ntgenogram of the chest revealed nothing abnormal. e blood pressure was 104 mm. of mercury systolic d 74 mm. diastolic. The patient was 47 inches (119 .) tall and weighed 49 pounds (22 kg.). Funduscopic amination revealed that the retinal arteries were nerally narrowed but that the narrowing was greater the right eye. Definite chronic sclerosis and spastic anges were not found. The arteries pulsated spon-neously; the pulsations were of the excursion type. merous exudates were noted in the macular and ripupillary portions of the retinas with a few peculiar aquelike exudates in both maculas and above the right k. The pigment had changed slightly in the maculas. e right disk was a little lighter than the left disk d slightly veiled. The nasal portion of the left disk as full. Both had exaggerated rings. The ophthal-ologist stated that all of these retinal changes were idual to previous edema, that no recent active changes re seen and that the patient was apparently doing markably well.

Reexamination on May 19, 1941.—The patient had en well except for bronchitis. She had had a pro-ductive cough and a fever of low grade in July and August 1940 and again during the three weeks before her admission to the clinic. She had not been absent from school on account of the cough. A roentgenogram of the chest and an examination of the sputum made elsewhere in August 1940 were said to have given negative results. Gain in weight had been steady since the patient's previous admission to the clinic. There had been no recurrence of headaches.

Examination revealed that the patient's blood pressure was 102 mm. systolic and 78 mm. diastolic. Her height was 50 inches (127 cm.), and her weight was 54 pounds (24.5 Kg.). There were many coarse rales in the left lower portion of the chest posteriorly. The patient expectorated about 4 teaspoons (16 cc.) of purulent material daily.

A roentgenogram of the chest revealed bronchiectasis in the posteromesial division of the bronchus leading to the lower lobe of the left lung. Bronchoscopic examination made on May 21, 1941 showed definite narrowing of this bronchus. This was dilated, and a considerable amount of secretion was obtained. A large amount of purulent secretion was seen coming from the left lower lobe bronchus. The right side appeared to be dry.

Examination of the ocular fundi at that time revealed generalized retinal arterial narrowing which was minimal to grade 1. Aside from an increased light reflex no hypertensive changes were visible. The disks were flat. There was a suggestion of pallor on the right. Changes in pigment were present in the maculas, and there was some retinal thickening. There were peripheral disturbances of the pigment owing to previous retinitis. There was no retinitis and no sign of activity of hypertension.

Operation on July 5, 1941—Removal of the lower lobe of the left lung for bronchiectasis was carried out by one of the surgeons at the clinic. The blood pressure before operation was 110 mm. systolic and 83 mm. diastolic; after operation it was 130 mm. systolic and 96 mm. diastolic. Operation was followed by uneventful convalescence, and the patient was dismissed from the hospital on the twenty-second postoperative day.

Reexamination on Dec. 16, 1941.—The patient was 51 inches (129.5 cm.) tall and weighed 57 pounds (26 kg.) at this time. The blood pressure was 108 mm. systolic and 70 mm. diastolic. A slight cough had persisted since the lobectomy, but this had become progressively less marked. In October 1941 (two months before this examination), the patient had had dysentery and had lost much weight. At this time she was regaining the lost weight rapidly, and her appetite was good.

The ocular fundi at this time revealed general narrowing of the retinal arteries, grade 1; there was no visible sclerosis or angiospastic lesions or scarring in the maculas. Some alterations in pigment, which were more marked on the right, were noted. There were a few small patches of exudate residual from edema above the right macula. The disks had exaggerated rings, and there was a slight increase in the amount of connective tissue on the right. Otherwise the ocular fundi were normal. The ophthalmologist stated that the recovery from the hypertensive changes continued to be excellent.

Reexamination on Sept. 1, 1944.—The patient had been rather well in the interval between October 1941 and September 1944. She had had measles and so-called "stomach influenza"; since June 1944 she had had a slight intermittent cough. She reported that

there had been slight, occasional headaches, which lasted for only a few minutes.

Examination revealed the patient's height to be 57 inches (145 cm.) and her weight 76 pounds (34.5 Kg.). The blood pressure was 106 mm. of mercury systolic and 70 mm. diastolic. The heart beat was forceful, and there were no audible murmurs. Examination of the chest, including a roentgenogram, revealed nothing of significance. The urine was normal, and the value for blood urea was 22 mg. per hundred cubic centimeters.

The ophthalmologist reported approximately as follows: Vision in the right eye was 6/10, 14/14 and in the left 6/12, 14/14. The pupils, reflexes and fields were normal. The fundi were essentially normal except for moderate scarring in the macular regions with some deposition of pigment and in the left eye a few hyalin-like deposits, a spot of pigment temporal to the right disk and a few slight changes in pigment in the peripheries. The disks were normal, with well defined margins. Slight glial thickening was noted at the poles, and an exaggerated choroid ring was present temporally on the left. The right disk was possibly slightly pale, and there was a slight increase of connective tissue in the cup. The arterioles were essentially normal. There was no visible sclerosis. The caliber of a few arterioles was perhaps a little small but was within normal li The ophthalmologist added: "I do not believe one c tell from the present retinal appearance that there ever been hypertension. There has been an exce recovery with no residual vascular damage."

SUMMARY

We have reported the case of a child w exhibited the features of malignant hyper sion, which was corrected by nephrecto Since the blood pressure has remained nor for five years, it is reasonable to conclude t the cure is permanent. The patient has b able to return to full activity and has even b able to withstand a major surgical proced without untoward effect. The observations this case suggest that the prognosis for malig hypertension in the cases of children who h unilateral atrophic pyelonephritis may be usually good, because the pathologic proce have not been of too long standing and he are reversible.

ACUTE GLOMERULAR NEPHRITIS IN AN INFANT WITH CONGENITAL SYPHILIS

JOSEPH YAMPOLSKY, M.D., AND DEWITT F. MULLINS JR., M.D.
ATLANTA, GA.

We present the following case with a report of autopsy and a review of the opinions of ious writers as to the diagnosis of nephritis he newborn and in young infants.

REPORT OF A CASE

. S. J., a Negro girl 2 months of age, was admitted Grady Hospital, Atlanta, Ga., May 26, 1942. Her f complaint was swelling of the hands, the feet, the nach and the face for the preceding five days and uent vomiting. Her weight on admission was 10 nds 10½ ounces (4,833.6 Gm.); her temperature 99.6 F. (rectal). The patient, who was a second d, had been delivered at home; she weighed 7 pounds 75.14 Gm.) at birth. She was breast fed. The her, according to her own statement, had shown ositive reaction to the Kahn test during her recent gnancy but had received no treatment.

hysical Examination.—On examination the child was nd to feel severe pain when touched. The legs med especially sensitive to touch. There was pro-nced pitting of the hands and feet. There was reme pallor of the conjunctivas, as well as of the cous membranes of the mouth. Nothing abnormal found in the heart or the lungs. The abdomen greatly distended. On the left side of the abdomen, ending from the costal margin to the iliac crest, re was a palpable mass, which was thought to be er the spleen or a polycystic kidney. The genitalia re normal. The extremities were apparently very nful on touch, with severe pitting edema and signs enlargement of the epiphyses of the long bones. The pression on examination was that this patient had following conditions: (1) congenital syphilis, syphilitic periostitis, (3) secondary anemia and (4) genital anomaly of the kidneys.

Course.—One day after the patient's admission 30 cc. citrated blood was given intravenously. Three days er the patient seemed to have difficulty in nursing, d the next day the child was apparently in great tress. Nikethamide was given, but the patient grew idly worse and died that afternoon.

Roentgen Examination.—Examination of the chest re-led no pathologic changes in the lungs or the heart. entgenographic examination of the long bones showed finite periostitis, osteochondritis and osteomyelitis.

Examination of the Blood.—Examination of the blood vealed a red cell count of 1,150,000, a white cell count 37,200 and a hemoglobin content of 6 Gm. per hun-d cubic centimeters of blood. Nothing abnormal was seen in the cells. The Kahn reaction was strongly positive.

Urinalysis.—Urinalysis showed orange-colored urine, which was cloudy. The reaction for albumin was 2 plus, and there were innumerable white blood cells and from 10 to 40 red blood cells per high power field. There were also innumerable granular and hyaline casts.

Preautopsy Diagnosis.—The diagnosis was as follows: (1) congenital syphilis, (2) syphilitic osteomyelitis, (3) syphilitic osteochondritis, (4) syphilitic periostitis, (5) syphilitic anemia secondary to infection and (6) syphilitic nephritis.

REPORT OF AUTOPSY

External Appearance.—The body was 21½ inches (54.61 cm.) in length and weighed 4,965 Gm. It was well developed and fairly well nourished and showed no anatomic abnormalities externally. The hair of the head was coarse, black and fairly abundant. Over the entire body there was edema, 2 to 3 plus; this edema was present over the face also. The conjunctivas were pale. The corneas were clear; the pupils of the eyes were regular and equal and measured 25 cm. in diameter. In the nose there was a large amount of grayish white mucoid material. The mucous membranes of the buccal cavity were pale, but not the tongue. The surface of the tongue was dry and brownish red. No superficial lymph nodes were palpable over the body. The neck was symmetric, and the trachea was in the midline. The thyroid was not palpable. The thorax was symmetric and infantile in type. The abdomen was full and symmetric and protruded above the level of the costal margin with the body in the supine position. The extremities were symmetric and well developed and presented a pitting edema, 2 to 3 plus. The palms and the soles showed a scaly, desquamative dermatitis. The nail beds were pale. Careful search of the corners of the mouth revealed no lesions.

Description of Internal Organs.—The panniculus adiposus was 1 cm. in thickness and was a pale yellowish gray. The subcutaneous tissues were noticeably edematous.

Peritoneal Cavity: The peritoneal surfaces were smooth and glistening. The peritoneal cavity contained approximately 150 cc. of clear, straw-colored fluid. The abdominal viscera were normally related.

Pleural Cavities: The pleural surfaces were smooth, shiny and translucent. Each pleural cavity contained 50 to 100 cc. of clear, straw-colored fluid.

Lungs: The lungs were normal in configuration and size. They retracted moderately as the pleural cavities were opened. The upper lobe of each lung and the middle lobe of the right lung were pale pinkish white

From the Department of Pediatrics and Pathology, nory University Medical School, and the Children's ard of Grady Hospital.

and spotted with hemorrhagic areas, averaging 5 cm. in diameter. The posterolateral portion of each lower lobe was dark pinkish red to reddish purple and was firm and normal in consistency. The cut surface was mottled bluish red and pinkish red; these darker areas stood out somewhat above the surface. The upper lobes and the middle lobe of the right lung were pinkish white on the cut surface; scattered throughout the parenchyma there were hemorrhagic areas, similar to those noted externally.

Pericardial Cavity: The pericardial surfaces were smooth and glistening. The pericardial cavity contained approximately 40 to 50 cc. of clear, straw-colored fluid.

Kidneys: The kidneys lay in a small amoun perirenal fat; they were moderately enlarged and a pale pinkish tan. The fetal lobulations were reta to a slight degree. The capsule stripped from e kidney completely and without difficulty. The exte surface of each kidney was smooth and pinkish t throughout this surface there were numerous pinp dark red areas, appearing to be tiny hemorrhages. the kidneys were sectioned, the cut edge rolled mo ately. The markings on the cut surfaces were va and cloudy. The strial markings stood out only fa well. There was no dilatation or inflammation of pelves or the calices. The ureter had its normal posi and showed no dilatation or tortuosity.

Fig. 1.—*A* and *B*, roentgenograms of upper extremities, revealing definite syphilitic periostitis, osteochondri and osteomyelitis. *C*, roentgenogram of lower extremities, revealing definite syphilitic periostitis, osteoch dritis and osteomyelitis.

Heart: The heart was of normal size and configuration. The myocardium was pale tannish brown. The endocardium was smooth and glistening. The valves of the heart were normal in appearance and size. The intima of the aorta was smooth and glistening. The coronary ostiums were patent.

Liver: The anterior edge of the liver lay 3 cm. below the costal margin and the right midclavicular line. The liver was normally related but appeared somewhat larger than normal; the capsule was transparent; both the external and the cut surface of the liver were pale pinkish brown and appeared meaty.

Spleen: The spleen was moderately enlarged; the capsule was stretched tightly, and the color was dark reddish purple. Section of this organ revealed the cut surface to be dark reddish purple. The malpighian corpuscles did not stand out.

Adrenal Glands: The adrenal glands were thin, pale and normally related. They presented no gross abnormalities other than their pallor.

Pancreas: The pancreas was normal in size, sha relationship and consistency. Both the external a the cut surface were a uniform pinkish white.

Retroperitoneal Structures: The areolar tissue lyi behind the peritoneum was edematous. This was tr also of the mesentery.

Microscopic Examination.—Lungs: The pleura w edematous and infiltrated with small round cells, plas cells, eosinophilis and a few polymorphonuclear leuk cytes. The alveolar walls were thickened, and t capillaries were greatly congested. The alveoli appear to be poorly aerated, and most of them contain albuminous material, red blood cells and large mo nuclear cells, some of which contained granules of bro pigment. Some of the alveoli also contained a fe polymorphonuclear cells. Many of the alveoli contain material which was apparently desquamated cells. T perivascular and peribronchial connective tissue w increased in amount and sparsely infiltrated with va

numbers of small round cells, polymorphonuclear ocytes and large mononuclear cells. Also, in the interstitial tissue there were focal and diffuse rations of similar cells. In addition to the cells ed, there were large mononuclear cells, which ared to be young myeloid cells. There were patchy ps of alveoli which were stuffed with red blood , a peculiar reddish blue fibrinous material and a polygonal cells with light blue nuclei, which ared to be connective tissue cells. There seemed e a generalized increase in the amount of inter- l connective tissue.

eart: The muscle fibers were moderately separated. striations stood out poorly. Between many of the cle fibers there were collections of small round cells cells which appeared to be young myeloid cells. perivascular connective tissue was edematous and trated with a few small round cells.

iver: The capsule was thin and uniform. Under- h it there was an occasional collection of small nd cells and an occasional eosinophil. The cell ls were somewhat irregular in size and shape. The plasm of many of the hepatic cells contained uoles. The sinusoids were dilated and contained ying numbers of nucleated red blood cells and large nonuclear cells, which appeared to be young myeloid s. The portal triads were infiltrated with small nd cells, plasma cells and an occasional eosinophil. e media and adventitia of some of the larger vessels re infiltrated with small round cells, plasma cells an occasional polymorphonuclear leukocyte and inophil.

Spleen: The capsule was thick and uniform. The alpighian corpuscles were not numerous; they were ied in size and irregular in shape. The sinusoids re packed with extremely large numbers of red blood ls. They also contained a fairly large number of lymorphonuclear cells and large mononuclear cells, hich appeared to be myeloid cells.

Adrenal Glands: The pericapsular fat and connective sue were infiltrated in a patchy manner with small nd cells and a few plasma cells. There were well capsulated islands of cortical cells in the capsules of th glands. In both the cortex and the medulla there re focal collections of small round cells and a few sma cells. The zona reticularis was vascular, and e cells were vesicular. There was a moderate amount medullary tissue in both glands.

Kidneys: The capsular line was somewhat wavy. here was no normal glomerulus in the section. Most the glomeruli showed a smudgy eosinophilic thicken- g and fibrosis, with adhesion to the capsule and ticeable crescent formation. A few had shrunken, ickened tufts. In many of the capsular spaces there re desquamated epithelial cells, leukocytes and fibrin. any of the tubules showed varying degrees of fibrosis, hile others were dilated and lined with low, atrophic ithelium and contained albuminous material. Through- t the section there were diffuse and focal infiltrations small round cells, plasma cells and a few polymor- onuclear leukocytes and eosinophils. Some of the naller collections of small round cells had extremely rk nuclei. Also, in the section there were small col- ctions of large mononuclear cells which had the appear- ance of young myeloid cells. The blood vessels were considerably congested.

Microscopic examination revealed the same picture in all sections from both kidneys. The cortical surface was irregular; beneath it were numerous dilated tubules containing pink-staining amorphous casts and lined with thin, atrophic epithelium. The epithelium of the proximal convoluted tubules showed granular degeneration with pyknosis and karyorrhexis. No normal glomeruli could be seen in any of the sections, but those located in the deeper portions of the cortex showed the most advanced lesions. Many of the superficially located glomeruli were increased in size, and the tuft filled the glomeruli cavity almost completely.

The endothelial cells lining the capillaries appeared swollen and contained large vesicular nuclei and abundant pink-staining cytoplasm. The basement membrane contained an increased amount of collagen, and the epithelial cells covering the glomerular capillaries appeared increased in number. The lumens of the glomerular capillaries were decreased in size, which resulted in an ischemic appearance, but some of them contained polymorphonuclear leukocytes. Many glomerular cavities contained several plasma cells and lymphocytes. The majority of the glomeruli showed pronounced thickening of the capsule, with formation of many epithelial crescents, and often the proliferation appeared to obliterate the glomerular tuft. Several glomeruli were completely hyalinized. The interstitial tissue was diffusely infiltrated with lymphocytes, plasma cells, some eosinophils and a moderate sprinkling of mononuclear cells, with occasional concentration around the more viable-appearing glomeruli. The larger arteries presented nothing abnormal, but the afferent glomerular arterioles showed subendothelial proliferation and a surrounding infiltrate, composed of plasma cells and lymphocytes. No spirochetes could be demonstrated in the sections with Dieterle's silver stain.

Pancreas: The lobules were well demarcated. The interlobular connective tissue was edematous and infiltrated sparsely with small round cells, plasma cells and a few polymorphonuclear leukocytes and eosinophils. There was a definite increase in the amount of interacinar connective tissue. Also, insinuated among the acini there were rather large collections of small round cells and eosinophils. The malpighian bodies were few but large, and their cells possessed nuclei which were darker than normal. The pancreatic ducts were not remarkable except for an infiltration of small round cells, plasma cells, polymorphonuclear leukocytes and eosinophils in the periductal connective tissue.

Lymph Nodes: The capsule was thin and intact. The cortical and medullary sinuses stood open and contained an occasional swollen, desquamated endothelial cell. No definite lymphoid follicles were present, the lymphoid tissue being arranged in thin sheets, among which were many cells undergoing karyorrhexis. The interstitial tissue was infiltrated sparsely with small round cells, plasma cells and a few eosinophils.

Thymus: The interlobular connective tissue was sparsely infiltrated with small round cells, plasma cells, a few eosinophils and a few large mononuclear cells, which appeared to be young myeloid cells. The corticomedullary demarcation was indistinct, and thymocytes were fewer than normal and showed much karyorrhexis.

There were a moderate number of Hassall bodies in the medulla, most of which showed cystic dilatation and contained fibrinous and leukocytic debris.

Sternum: There was an occasional collection of small round cells and eosinophils in the periosteum. Only a small amount of marrow was present, and this showed noticeable hyperplasia.

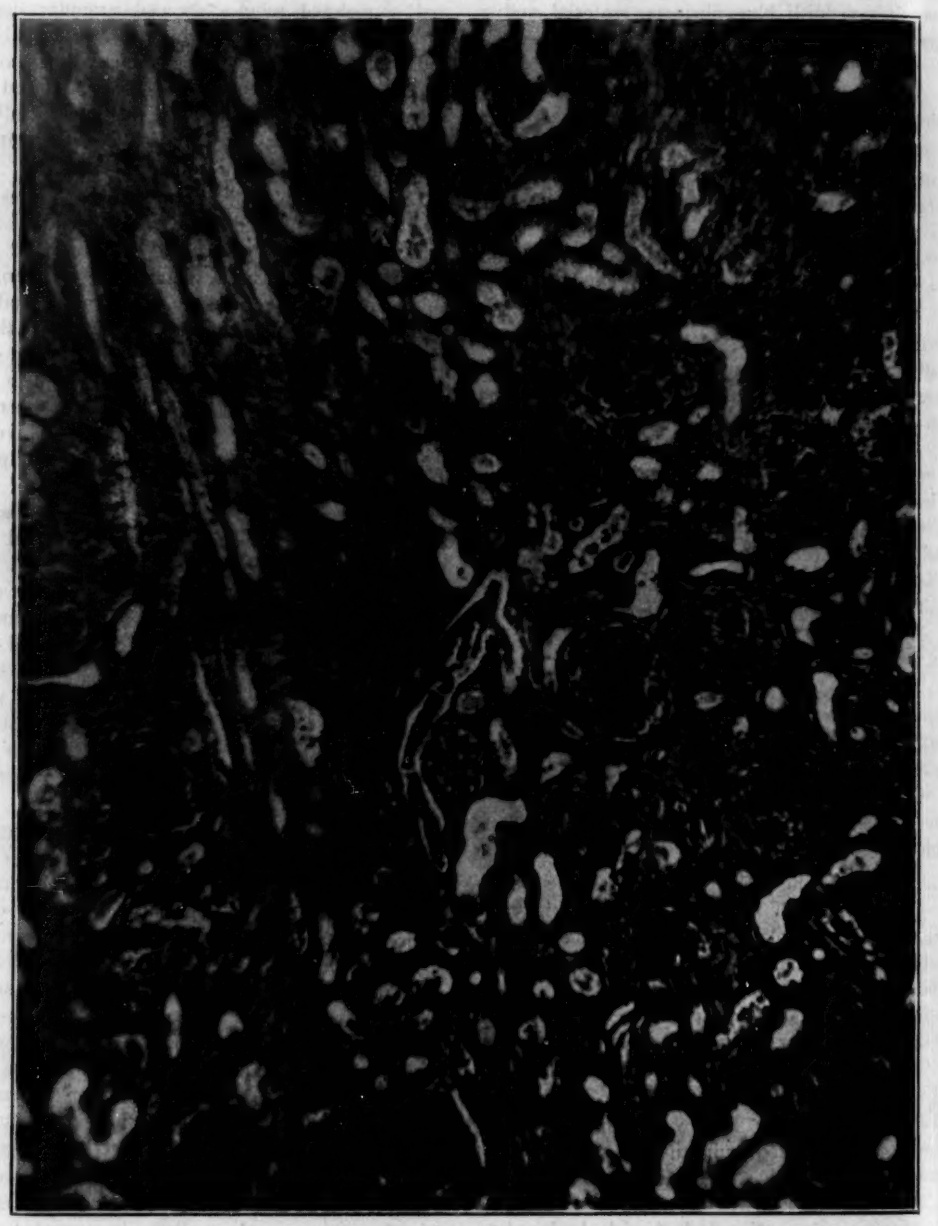


Fig. 2.—General view of cortex, showing dilatation of tubules with casts and glomerular lesions in a kidney.

Study of all organs stained with Dieterle's stain for spirochetes failed to reveal the presence of these organisms.

Anatomic Diagnosis.—The following anatomic diagnosis was made: (1) anasarca; (2) large, pale, cloudy kidneys with multiple pinpoint hemorrhagic areas, of undetermined cause; (3) bronchopneumonia of the lower lobes of both lungs and multiple small hemorrhages of the other lobes; (4) anemia; (5) hepatomegaly; (6) splenomegaly, and (7) desquamative dermatitis of palms and the soles.

Microscopic Diagnosis.—The following microscopic diagnosis was made. (1) passive congestion of lungs with early bronchopneumonia; (2) chronic inflammation of all the organs; (3) extensive ... stage glomerulonephritis, and (4) hyperplasia of bone marrow.

REVIEW OF THE LITERATURE

A review of the literature will show that authorities differ as to whether this condition exists and also as to the diagnosis and the prognosis in hemorrhagic nephritis in the young.

interesting case has been studied by ito Santo and Mauricio Correia.[1] They ibed the case of a boy 3 months of age. hief complaint began about a week previous s admission to the hospital. The mother noted that the baby urinated little and that canty urine was of a bloody color. Urinaly- and granular casts. The mother admitted that she had what she called a "marital affection." During her recent pregnancy she had received twenty-one injections of some drug, but apparently it was not antisyphilitic.

On examination the patient revealed marked emaciation; there was chronic coryza, the respi-

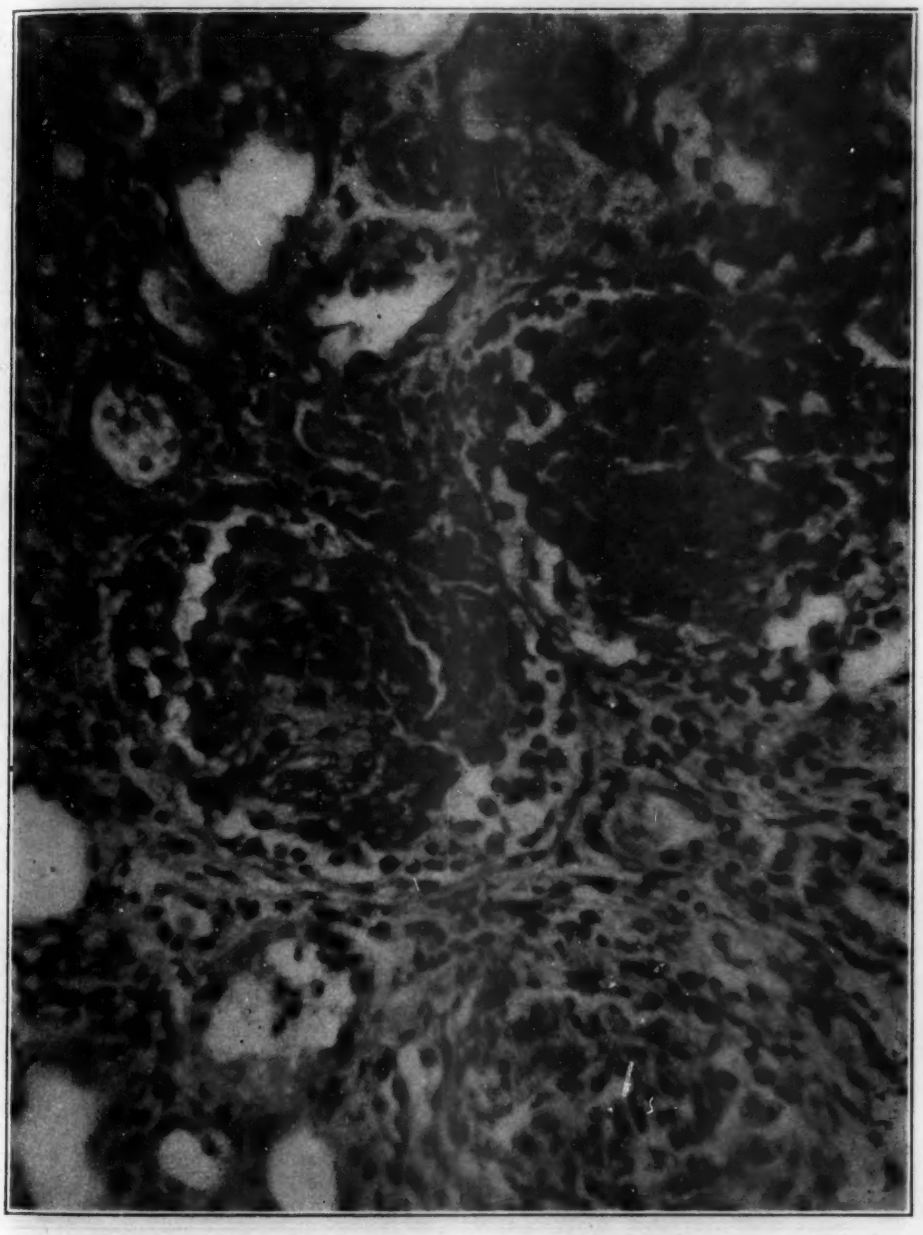


Fig. 3.—View of glomerular lesions in a kidney, showing thickening of basement membranes, plasma cells and phocytes in glomerular cavity and around capsules.

showed a high albumin content and no sugar. e sediment contained a considerable number red blood cells and a large number of hyaline rations were harsh and the nose was saddle-like. The skin around the anus presented typical syphilitic ulcerations. The facies were characteristic of congenital syphilis. The inguinal, epitrochlear and laterocervical lymphatic glands were infarcted. The patient had generalized edema. The lower margin of the liver was

1. Espírito Santo, C. A., ad Mauricio Correia, J.: Case of Nephritis, Probably of Syphilitic Origin, in Three Month Old Baby; Cure, Pediat. prát., São Paulo **12**:27-32 (Jan.-Feb.) 1941.

2 fingerbreadths below the costal arch, and it was hard. There was pronounced periostitis on the external side of each femur and the inner side of each tibia. The spleen was palpable and hard.

Several urinalyses revealed an abundance of albumin and sediment containing a number of red blood cells and a small number of leukocytes. On roentgenographic examination the visibility of the kidneys was obstructed by the shadow of the intestines, which were distended with gas. No abnormal shadows were seen in the region of the ureters or of the urinary bladder. The Wassermann test gave a strongly positive reaction. This patient was given syphilitic treatment and apparently recovered.

In summing up this case the authors commented on the frequency of renal disease in infants with congenital syphilis. In reviewing the literature they were able to demonstrate that renal and hepatic diseases occur frequently and that about 40 per cent of the infants who have renal diseases are syphilitic. They also state the opinion that considerable improvement can be seen in children with renal disease when antisyphilitic treatment is used. They expressed the opinion that their results have been corroborated by Hintzelam, Lang, Reuss and Hutinel.

Fig. 4.—Thickening of glomerular capsule of a kidney, with formation of epithelial crescents.

other interesting report on a study of routine microscopic examinations of the kidneys in born and newborn infants under 14 months age was made by Friedman, Grayzel and Lederer.[2] They stressed the fact that in textbooks and reference works on pathology little mention is made of such lesions and that the literature contains surprisingly few contributions on this field. They called special attention to a number of cases which have been studied by pediatricians and urologists. For instance, in 1908 Karsner[3] described a case of congenital acute nephritis in an infant who died less than one hour after birth. Three cases of neonatal nephritis were described by Conrad,[4] and special attention is called to Fishberg's case[5] of an infant 4½ months old who died from subacute glo-

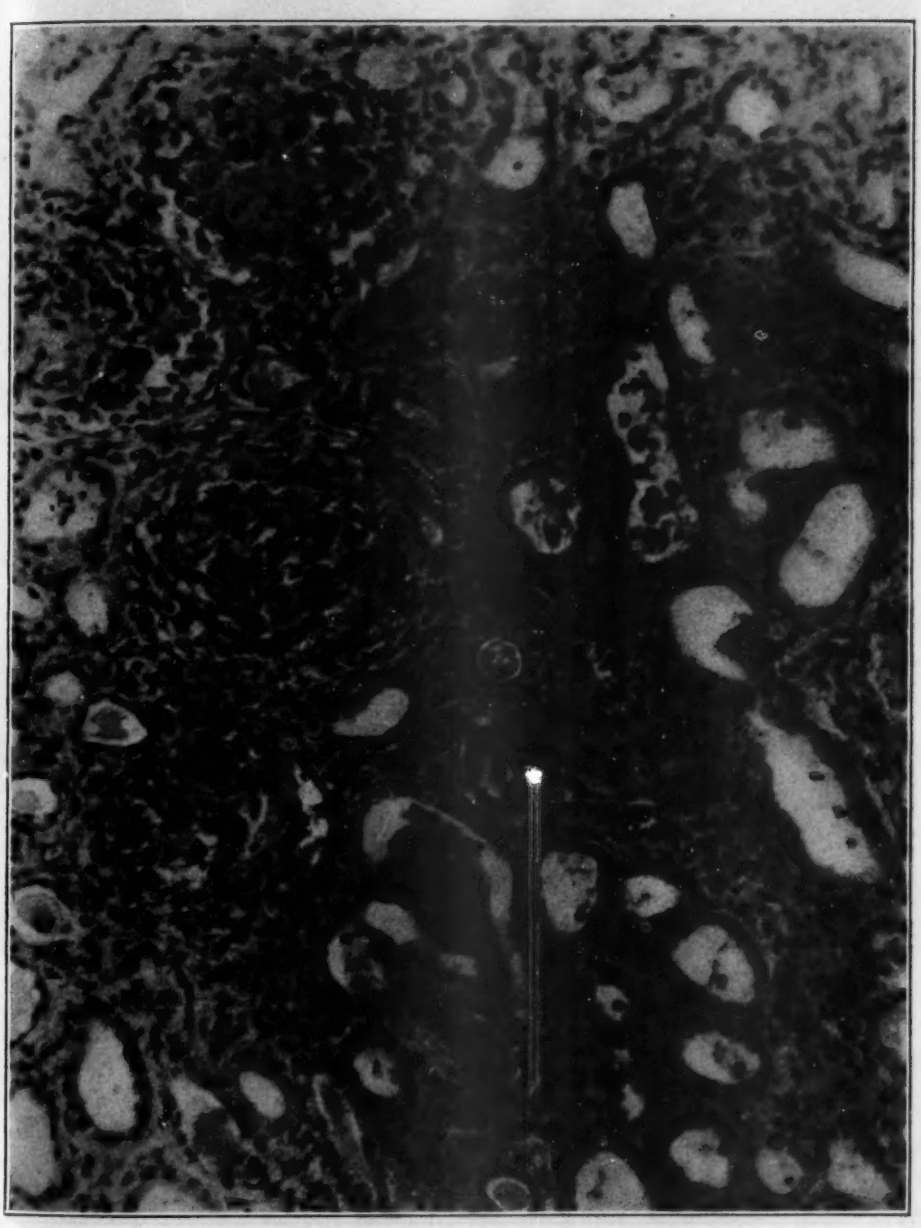


Fig. 5.—Widening cell involvement of glomeruli of a kidney, with infiltration of lymphocytes and plasma cells in interstitial tissue.

2. Friedman, H. H.; Grayzel, D. M., and Lederer, F.: Kidney Lesions in Stillborn and Newborn Infants, Arch. Path. **18**:699-713, 1942.

3. Karsner, H. T.: Congenital Nephritis, New York M. J. **88**:1076-1079, 1908.

4. Conrad, C. E.: Urinary Findings in the Newborn: Report of Three Cases of Neonatal Nephritis, South. M. J. **31**:636-640, 1938.

5. Fishberg, A. M.: Hypertension and Nephritis, ed. 4, Philadelphia, Lea & Febiger, 1939, p. 435.

merulonephritis and whose kidney was studied for the pathologic changes.

Friedman, Grayzel and Lederer had an opportunity of doing routine microscopic examinations of the kidneys of 13 stillborn and newborn infants. Recently they have studied 100 consecutive stillborn and newborn infants under

showed anasarca, hydrothorax and ascites, w
could be ascribed to renal damage. Some sh
only edema of the extremities. Of the 10 pa
whose urine was examined, only 2 pa
showed occasional red blood cells, and 5
albuminuria, which the authors stated
attributable to other causes, such as fever.

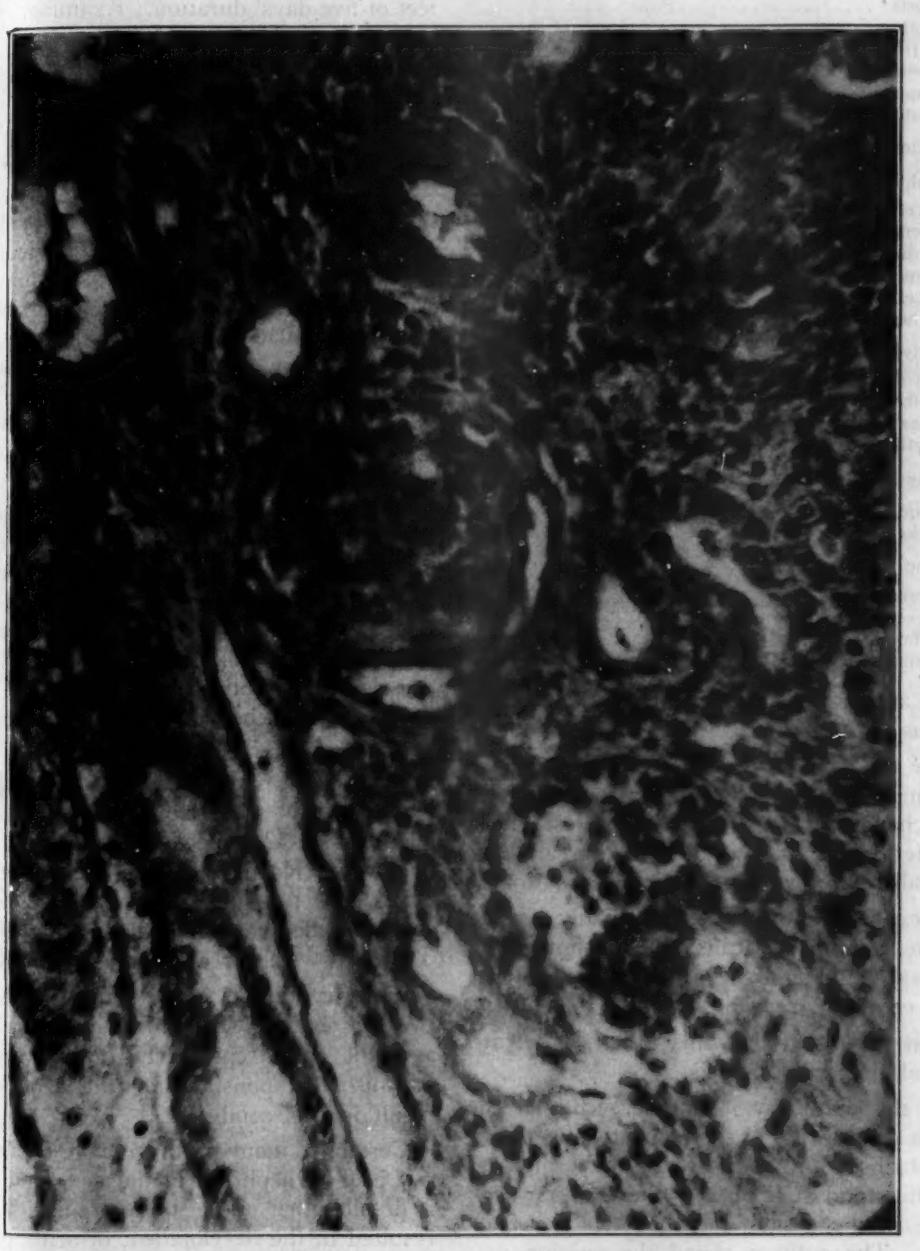
Fig. 6.—Highly magnified glomerulus from a kidney, forming epithelial crescents.

14 months of age and found 17 more examples of the lesions which they had described. The clinical features of the cases referred to are few. The average age of the infants whose kidneys were studied was 2.7 months. There were 4 stillborn infants in the group, and none of the children was less than 3 weeks of age. Some

As to the causation of this condition, not
definite was established by these writers as
as the maternal factors are concerned. T
was no high incidence of maternal infecti
Five of the mothers had syphilis, latent, a
or cured. Only 2 of the children of t
mothers showed positive reactions to serol

and in only 1 of these children was there nce of congenital syphilis at necropsy. The child died from hemorrhagic encephalitis ving arsenical therapy. Friedman and his iates expressed the view that theoretically infectious, hormonal or other maternal development of the kidneys, but he did not mention the vascular changes observed in these patients.

As to the pathologic changes involved, these authors expressed the conviction that the alterations in the blood vessels are the significant



g. 7.—Endothelial proliferation and perivascular infiltration of a kidney with lymphocytes and plasma cells.

rs acting on the fetus in utero may be onsible for the lesion.

nese authors stated the opinion that the ns in the kidneys may be developmental, mmatory, degenerative or vascular in origin. cheimer [6] expressed the view that this condi- in newborn infants is probably noninflam- ry in origin. He attributed it to retarded etiologic factors. They stated that the cause of these changes is still problematic.

There follows a list of associated pathologic conditions which they found in many infants

6. Herxheimer, G.: Ueber hyaline Glomeruli der Neugeborenen und Säuglinge, Frankfurt. Ztschr. f. Path. **2**:138-152, 1909.

with nephritis, together with the number of infants involved:

Condition	No. of Infants
Infectious diseases	
Meningitis	3
Abscesses of skin	2
Sepsis	2
Peritonitis	1
Otitis media	3
Pneumonia	8
Empyema	1
Acute bacterial endocarditis	1
Anomalies of the heart and great vessels	5
Cerebral lesions	
Porencephaly	1
Anencephaly	1
Hydrocephalus	1
Hemorrhagic encephalitis	1
Laceration of tentorium cerebelli with hemorrhage	1
Blood dyscrasias	
Thrombopenia	1
Leukemic myelosis	1
Unexplained widespread hemorrhage	1
Erythroblastosis	1
Anomaly of a kidney	1
Metabolic disorders	
Rickets	2
Obstetric abnormalities	
Prematurity	2
Breech presentation	1
Difficult labor	1
Premature separation of placenta	1

These authors briefly summarized their observations as follows:

1. The lesions involve the arterioles and their appertaining glomeruli and are associated with hyaline changes in the tufts and capsules. They are bilateral and focal in distribution and are recognizable only by microscopic examination.

2. They are not associated with any clinical syndrome, nor is their causation or significance understood, although they are congenital and may be of vascular origin.

3. It is suggested that this lesion be termed congenital glomerulosclerosis.

From this summary it can be seen that the causation of nephritis in the newborn is not definitely established, although some of the mothers had syphilis and at least 2 of the children born of these mothers showed a positive serologic reaction. The authors stated the opinion that the lesions observed in their patients may be the result of toxic, infectious, hormonal or other maternal factors, which act on the fetus in utero.

SUMMARY

It is difficult to place the case reported in any of the categories described by Frie A Negro girl 2 months of age was adm to the hospital with the chief complaint of s ing of the hands, the face, the stomach an feet of five days' duration. Examination sh signs of congenital syphilis, such as enla spleen and liver and marked periostitis, there was a maternal history of syphilis. patient lived but a short time. Micros examination of the kidneys revealed the lowing changes:

There were no normal glomeruli in the se examined. Most of the glomeruli demonstr a smudgy eosinophilic thickening and fibr with adhesion to the capsule and conside crescent formation. In many of the cap spaces there were desquamated epithelial leukocytes and fibrin. Many of the tu showed varying degrees of fibrosis, while o were dilated and lined with low, atrophic thelium and contained albuminous mat Throughout the section there was also di and focal infiltration of small round plasma cells and a few polymorphonuclear and eosinophils. There were also small c tions of large mononuclear cells which ha appearance of young myeloid cells. The l vessels were noticeably congested. No rochetes could be demonstrated in any o sections.

Repeated urinalyses showed orange-co urine, which was cloudy; the reaction albumin was 2 plus, and there were innu able white blood cells and from 10 to 40 blood cells per high power field. There also innumerable granular and hyaline c The child's Kahn reaction was strongly pos

Many conditions cause hemorrhagic neph in the young. The most common etiol agent is the streptococcus or the many o organisms responsible for the sequelae w result in this condition.

Certainly, many factors active in uterine can lead to nephritis in the very young, a is possible that one of these predisposing fac resulted in the development of acute glome nephritis in this child with congenital syph

Suite 311, Doctors Building.
Grady Hospital.

ESOPHAGEAL STENOSIS DUE TO LACTIC ACID

JOSEPH B. TRAINER, M.S.; WILLIAM W. KRIPPAEHNE, B.S.;
WARREN C. HUNTER, M.D., AND DANIEL A. LAGOZZINO, M.D.
PORTLAND, ORE.

The recent report of Young and Smith [1] has ed attention to the possible caustic effect of ic acid. These writers reported 3 deaths to the action of this substance in feeding nulas and supported their observations with derived from experiments with rabbits. possibility that severe reactions may occur nfants as a result of errors in preparation eeding formulas seems to have been gen- ly overlooked in recent years, despite the espread use of lactic acid. While Young Smith were unable to find mention of this stance in any of the standard textbooks on icology, we found it briefly discussed under n "Caustic Acids" and "Organic Acids" in lmann's "Manual of Pharmacology." [2]

We have recently encountered a case exhibit- the chronic effects of accidental swallowing his substance. So far as we have been able scertain, the late sequelae of lactic acid in the nentary tract have never been recorded. A iographic search brought to light one refer- e worthy of being added to the literature cited Young and Smith. It is especially interesting ause it appears to be the first record of exten- e use of lactic acid by clinicians as a thera- tic agent. Eckerbom [3] reviewed a presenta- n of the subject by Dr. Jelinek at the ctoren-Collegium in Vienna, Nov. 9, 1885, ether with a discussion by Bum, Mosetig- orhof, Wienlechner and several others who l made use of this substance. Jelinek's descrip- n of the action of lactic acid is interesting.

When fresh mucous membrane is painted with lactic l there immediately appear marked reddening, and n even thickening, of the epithelium, but there is er pain or formation of a crust. If, however, patho- ically changed mucosa is treated, an intense burning n occurs, and a corrosion body is formed. The re watery and edematous the swelling, the more nounced is this reaction, and the more painful the nted part may be. If it is located in the larynx, rseness and cramp of the glottis follow if the solu- n applied is as strong as 20:100. However, the reaction is gradually reduced in degree under the treatment described, and eventually concentrated lactic acid can be used, even in the larynx.[3a]

Bum described the use of lactic acid–silica paste, with which ". . . clearly pathologic, spongelike, easily bleeding granulations change to grayish black, easily removed, porridge-like masses; after this change the wounds appear as if scraped out with a sharp instrument. Their walls appear reddish, feel firm and do not bleed even if roughly handled." Mosetig-Moorhof used up to "1 syringe of 50 per cent solution" in treating subcutaneous epithelioma and obtained "good results." Up to that time these men had used lactic acid as a therapeutic agent as follows: Jelinek had used it in the treatment of laryngeal tuberculosis; gangrenous, granular and hypertrophic pharyngitides; ozena, and scrofulous and hypertrophic rhinitis; Bum, in the treatment of localized tuberculosis of the skin and of the lymph glands with ulceration and fistulas; and Mosetig-Moorhof, in the treatment of lupus, papilloma, epithelioma and carcinoma.

In the German pharmacopeia of Eckerbom's day (1886) lactic acid is described as *Acideum lactecium*, a colored, inodorous, clear and somewhat thick-flowing fluid, not vaporizing at ordinary temperatures. A current description of lactic acid is found in the "Pharmacopeia of the United States" [4] and the "British Pharmacopeia." [5] In the former it is listed as "a mixture of $HC_3H_5O_3$ and lactic anhydride equivalent to a total of not less than 85 per cent and not more than 90 per cent of $HC_3H_5O_3$." Lange's "Handbook of Chemistry" [6] gives the dissociation constant of lactic acid as 1.55×10^{-4}. A 10 per cent solution is roughly equivalent to a one and one-tenth molar solution. Calculated by the usual formula the p_H of such a solution is 1.88. However, the ordinary method of calculation is not strictly applicable when the molar strength is as high as in the present instance; so the p_H values of a series of dilutions of lactic acid

From the Departments of Pathology and Pediatrics, iversity of Oregon Medical School.

1. Young, E. G., and Smith, R. P.: Lactic Acid, Corrosive Poison, J. A. M. A. **125**:1179-1181 (Aug. 1944.

2. Sollmann, T.: A Manual of Pharmacology, ed. 6, iladelphia, W. B. Saunders Company, 1942.

3. Eckerbom: Lactic Acid as a Caustic, Eira **10**: 17, 1886.

3a. Prof. Olof Larsell provided a translation from the Swedish.

4. Lactic Acid, in Pharmacopeia of the United States XII, Easton, Pa., Mack Printing Co., 1942, p. 23.

5. Lactic Acid, in British Pharmacopeia, London, Constable & Co., Ltd., 1932, p. 26.

6. Lange, N. A.: Handbook of Chemistry, ed. 3, Sandusky, Ohio, Handbook Publishers, Inc., 1939.

U. S. P. were determined electrometrically by one of us (J. B. T.). Similar dilutions in reconstituted evaporated milk, such as is used in preparation of feeding formulas, indicated that a 20 per cent solution has a p_H sufficiently low to act as a direct caustic agent, while an aqueous solution of only 10 per cent has that property (fig. 1).

These data emphasize the fact that the purely chemical concept of lactic acid as a weak acid should not be carried over to physiologic thinking. They indicate the basis of its caustic activity, which, taken in conjunction with the case to be reported here and the observations of Young and Smith, makes it evident that lactic acid is a potentially dangerous substance.

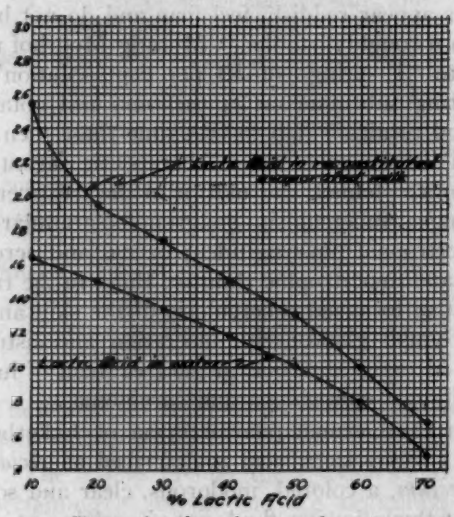


Fig. 1.—Chart showing the p_H of various solutions of lactic acid U. S. P. in reconstituted evaporated milk and in water.

REPORT OF A CASE

History.—J. H., a month old boy weighing 6.7 Kg., entered Doernbecher Memorial Hospital for Children, Portland, Ore., on Aug. 26, 1944 with a history of abrupt onset of inability to swallow, with regurgitation of his feeding formula into the nose and mouth for the previous twenty-four hours. His history revealed that at 2 weeks of age the infant received by error a teaspoon of lactic acid of full strength (U. S. P. 87.5 per cent), instead of elixir of phenobarbital. Ingestion of the acid resulted in visible burns in the oral cavity, with stasis of secretions in the pharynx. Because of burns of the mouth the child required hospitalization for ten days. Convalescence was complicated by an attack of measles followed by otitis media, which persisted for a month. Stasis in the pharynx required postural drainage before the child could be fed, and even then there were gagging and regurgitation. At 3 months of age he could tolerate full feedings of the formula but could not swallow solid food which was added to them. The patient was otherwise asymptomatic until he reached 5 months of age, when inability to swallow suddenly developed. This condition lasted five hours and was relieved by vomiting a small piece of paper. Otherwise the infant did well until the final episode, when he again could not swallow feedings and regurgitated through the nose and m The parents stated that the baby appeared h was afebrile and was in full control of his extrem

Physical Examination. — Physical examination vealed an irritable, mildly dehydrated 7 month old who did not appear to be in acute distress. His perature was 102.4 F. The pharynx was injected was filled with frothy mucus. The infant gagged offered water and regurgitated it through the nose mouth. The chest was resonant throughout; expa was free and equal; numerous resonating rales o sizes were heard throughout the pulmonary fields, ciated with rough breath sounds. No neuromus irregularities were noted. Hematologic examin revealed a white cell count of 8,000, with polymor nuclear leukocytes 68 per cent, staff forms 2 per small lymphocytes 27 per cent and monocytes 3 cent. A fluoroscopic examination made while the swallowed barium sulfate revealed an obstruction acteristic of stricture at the level of the tracheal b cation. Esophagoscopic examination showed an scarlike stricture, which had a small slit opening.

Diagnosis, Treatment and Course.—The impres was that the infant had an esophageal stricture d an old corrosive burn, complicated by aspiration p monia. The child was given sulfonamide compou fluids administered parenterally and supportive tr ment but failed to respond; he died on his second in the hospital, August 27.

REPORT OF NECROPSY

Gross Examination.—Only the pertinent observa need be described. The body was 65 cm. in length apparently well nourished. The anterior fontanel open, the posterior closed. The mucosa of the cavity was smooth, pink and glistening. There no visible teeth. The subcutaneous fat was from 5 mm. thick anteriorly.

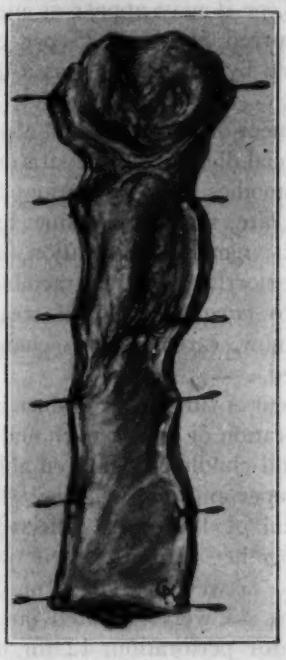
The opened esophagus (fig. 2) exhibited a stric just below the level of the thyroid cartilage. lumen had a circumference of 2 cm. above the s ture; it narrowed to a little less than 1 cm. at stricture and broadened to 2 cm. below it. A se stricture was located 2.4 cm. inferiorly; at this p the opening again narrowed to approximately 1 A small wad of folded paper was removed from region between the strictures. The paper was m but still somewhat firm. A perforation of the es agus 2 mm. in diameter was present midway bet the strictures and appeared to extend posteriorly the mediastinum. There was a green discoloration the mediastinal tissues above and below the perf tion for a distance of approximately 5 mm. B the second constriction the circumference of the es agus broadened to 2.4 cm.; it was 3 cm. at the car orifice.

The mucosa of the trachea and the main bro was pink, with a red exudate on its surface. upper lobe of the left lung was crepitant; the lo lobe was firm and red posteriorly and crepitant pink anteriorly. The right lung was pink both e nally and on the cut surface, and numerous petec were scattered over the external surface. The ga mucosa had a few small areas of focal hyperemia was otherwise without gross change.

After thorough fixation the esophagus was secti Sections in the longitudinal direction showed a dis whitish line 2 to 3 mm. thick in the presumptive cosal layer at the level of the stricture. Elsew

mucosa was less than 1 mm. thick. In the loca- of the submucosa all along this area a yellow to line was to be seen. The muscularis was not defined macroscopically. A transverse cut that led the small opening in the mucosa previously oned readily explains the longitudinal ridge that d so plainly from the inside of the opened esoph- The perforation extended at an angle through all for a total distance of 2 cm. in the longitudinal ion, with a breadth of 1 cm. in the transverse ion. The outer part of the esophageal wall was mushy and blackish.

croscopic Examination.—Esophagus: Sections of s cut longitudinally at, above and below the point nstriction disclosed a transitional stratified squa- epithelium that was not appreciably thicker than al or irregular in outline save for a dipping into



g. 2.—Halftone drawing of the esophagus, natural The upper stricture is the more pronounced. ween the strictures is a prominent ridge, over which small defect leading into the esophageal wall. mmation and necrosis are responsible for the ing.

. The various layers normal to this type of osa were represented in the proper order. At one t a microscopic-sized denuded area interrupted the mous surface. The connective tissue here showed ence of necrosis or digestion, but there was no mmation. There was no way of knowing whether not this condition was the result of the digestive n of vomitus. The submucosa was considerably kened, especially where the greatest stenosis existed. thermore, the density of the tissue here and its ndant collagen contrasted with the looseness of the mal structure of the submucosa. Rather deep down a small, elongated focus of hemorrhage. There was an increase of connective tissue in the muscularis, re the tissue was seen in the form of distinct aks. One section passed through the small point erforation of the mucosa. Here was seen not only rosis of the mucosa and the submucosa but also defined, acute, exudative inflammation, which spread into the submucosa and the muscularis. Most of the wandering cells were neutrophilic granulocytes, the remainder being monocytes and lymphocytes.

Lungs: The bronchi showed numerous areas containing both neutrophils and desquamated epithelium. Some air sacs were likewise filled with granulocytes. In both bronchi and air sacs there was occasional granular bluish material, such as one associates with vomitus, together with masses of yellowish pigment which might have been bile. It seems likely, therefore, that the bronchitis and the pneumonia had an aspiratory basis. Atelectasis and hyperemia completed the picture.

COMMENT

The facts of the present case require little elaboration. The clinical history was exceptionally clearcut and was in accordance with the observations at the postmortem examination, which was conducted within an hour after the patient's death. There was a history of sudden onset of caustic poisoning, with esophageal stenosis which developed steadily and was made suddenly acute by lodgment of a foreign body (paper wad) between two strictures. This incident may or may not have been associated with the existence of the small area of perforation. The presence of the foreign body, in all probability, led to the final episode of regurgitation, aspiration pneumonia, and asphyxia.

One may say, then, that poisoning with lactic acid initiates a series of events which are disabling at best; at worst, as in this case, the poisoning is an essential contributory cause of death. The present case differed from the cases of acute poisoning by lactic acid which have already been cited, but the pathologic process was undoubtedly the same in all. The baby's survival of the immediate effect of the caustic is ascribable to the fact that spasm and regurgitation of the bulk of the acid took place almost immediately, so that little if any acid reached the stomach or bowel and was absorbed.

SUMMARY AND CONCLUSIONS

An infant had caustic burns of the mouth and pharynx because of ingestion of concentrated lactic acid. His case differed from those previously reported in that the patient lived long enough for healing and esophageal stricture to take place.

The caustic effects of lactic acid described by Jelinek and Bum in 1885 are essentially similar to the changes observed in the present case.

The thesis of Young and Smith, that concentrated lactic acid should be considered a caustic poison and that care should be exercised in labeling and dispensing this substance, is strongly supported by the facts of the present case.

University of Oregon Medical School.

Progress in Pediatrics

GASTRIC AND DUODENAL ULCERS IN INFANCY AND IN CHILDHO

EDWARD J. DONOVAN, M.D., AND THOMAS V. SANTULLI, M.D.
NEW YORK

This report is based on a study of cases of gastric and duodenal ulcers encountered at the Babies Hospital from 1930 to 1944; it includes a presentation of the case of a 12 year old boy who was operated on for duodenal ulceration with obstruction. The age group covered is from birth to 12 years.

Although peptic ulcer is rarely demonstrated in infancy and childhood, it probably occurs with greater frequency than is reported. Of the 8,260 cases of peptic ulcer listed in the records of the Mayo Clinic from 1906 to 1924 Proctor [1] found only 2 involving children. However, he emphasized the fact that the history of ulcer often dates back to childhood, when the condition existed unrecognized.

Duodenal ulcer is more common than gastric ulcer, the ratio varying from 2:1 to 6:1 in different reports.

Ulceration has been reported to occur in utero. It probably accounts for some cases of melena neonatorum. In the newborn, as was pointed out by Kennedy,[2] the course is acute, and the lesion either heals rapidly or causes sudden, severe and generally fatal hemorrhage. The diagnosis is usually made at necropsy. In infants the clinical picture is obscure, and hemorrhage seems to be the dominant feature. For patients who have reached the age of 6 or 7 years the diagnosis is more easily made. The symptom complex is sometimes as clear as that in adults, but more frequently it is atypical. Peptic ulcer should be suspected in all cases of indefinite abdominal pain in children, particularly if the pain is epigastric. Proctor [1] stated, "The most important single factor in the diagnosis is realization that chronic peptic ulcer occurs in children." Roentgenologic studies may reveal the lesion, but it is not so easily demonstrable as in adults. Gastric analyses are not very practical, and the results for children are not so significant as for adults. The symptoms often remain unrecognized for a considerable period, until perforation or sten occurs.

According to Proctor,[1] bleeding is presen about 40 per cent of the cases; it varies fro small amount of blood in the vomitus to pro hematemesis, or it may appear as melena. P foration occurred in about 25 per cent of cases reported by him; there was a mortalit 50 per cent.

The treatment of uncomplicated ulcers is m cal, with special diet and administration of alk and antispasmodics. The indications for surg intervention are much the same for child as for adults, namely, perforation, obstruct recurrent hemorrhage and intractable pain responsive to adequate medical treatment. far as we know, carcinomatous change has been reported.

The problem of surgical procedure in the tr ment of ulceration of the stomach and duoden in infancy and childhood has been ably revie by Bird, Limper and Mayer.[3] In 1941 they p sented a total of 119 cases collected from literature, together with 1 of their own. Of 119 patients, between the ages of 34½ ho and 15 years, 53 were operated on for pylo stenosis, 43 for perforation, 12 for hemorrh and 11 for uncontrollable symptoms. The stom was involved in 30 patients and the duoden in 83; in 5 others there were multiple lesio involving more than one part. In 1 patient th was an ulcer in an accessory pouch of an ano alous stomach. Fifty-two gastroenterostom and 3 pyloroplasties were accomplished, with fatalities. There were 17 resections in all, w 2 deaths.

Ladd and Gross [4] reported the case of an year old boy with a history of vomiting and l of weight of two years' duration. Roentge logic examination showed pyloric obstructi with 50 per cent gastric retention at the end

From the Babies Hospital.

1. Proctor, O. S.: Peptic Ulcer in Childhood, Surg., Gynec. & Obst. **41**:63, 1925.

2. Kennedy, R. L. J.: Peptic Ulcer in Children, J. Pediat. **2**:641, 1933.

3. Bird, C. E.; Limper, M. A., and Mayer, J. Surgery in Ulceration of Stomach and Duodenum Infants and Children, Ann. Surg. **114**:526, 1941.

4. Ladd, W. E., and Gross, R. E.: Abdominal S gery in Infancy and Childhood, Philadelphia, W. Saunders Company, 1941. p. 19.

een hours. A posterior gastroenterostomy performed for a cicatrizing duodenal ulcer. years later the boy had gained 33 pounds Kg.) and had no symptoms.

ogan and Walters [5] presented the case of a ear old girl who complained of abdominal vomiting and melena, in whom roentgenohic examination disclosed an ulcer crater cm. in diameter on the lesser curvature of stomach. Surgical intervention was undern because of the hemorrhagic nature of the n and because of suspicion of malignancy. gmental excision of a portion of the stomach, ding the ulcer, was made with the electroery. Pathologic examination showed a sube hemorrhagic gastric ulcer, 2 by 1 by 5 The patient made an uneventful recovery. ostoperative gastrojejunal ulcer occurs in ren, as it does in adults. Strode [6] reported his complication.

the present report no attempt will be made lassify the ulcers. The cases are presented hronologic order. It is to be noted that the was the primary pathologic condition in the 10 cases reviewed, including the case h is reported in detail. In the other 3 cases ulcer was not the principal lesion.

VIEW OF CASES FROM BABIES HOSPITAL

SE 1.—C. C., a 22 month old girl, was admitted abies Hospital on Jan. 26, 1930, with a history ever and vomiting for the preceding thirty-six s. The vomitus was described as "watery" and ot containing any blood or fecal material. There been no bowel movements for two days. Subset questioning of the father revealed that three or days before her admission the child was playing a glass bottle, which broke. When the parents to assemble the pieces, they found that a small on was missing. It was their belief that she t have swallowed this missing piece of glass. e was no history of any previous gastrointestinal rbance.

xamination revealed that the child was acutely ill; rations were labored and grunting but not rapid. legs were drawn up at the hips. The temperature 101 F., the pulse rate was 200 and the respiratory was 24. The abdomen was full and distended, small dilated veins visible over the upper part. e were signs of fluid in both flanks. Rectal examon revealed nothing abnormal. An enema admined on the child's admission gave a small fecal n. A diagnosis of peritonitis with possible rupd viscus was made.

aparotomy was performed with local anesthesia. n the peritoneal cavity was opened, a quantity ir together with about 2,500 cc. of dark brown fluid escaped. The fluid contained particles of food, curds of milk and globules of oil. During the exploration the child ceased breathing. The anterior wall of the stomach was opened, and a perforation about 3 cm. in diameter was observed on the posterior wall of the fundus. The edges were ragged, soft, frayed and deeply hemorrhagic. No foreign body was encountered during the surgical procedure, and none was found later by the medical examiner. Sections cut from the edges of the perforation revealed necrosis of the mucosa and edema of the submucosa, in which lay several thrombosed vessels. The muscular coats were edematous and infiltrated with polymorphonuclear leukocytes. The following pathologic diagnosis was made: "acute ulcer with secondary inflammation of the gastric wall, although the presence of a previous gastric lesion cannot be excluded."

Case 1 was previously reported by Dr. B. R. Shore.[7]

CASE 2.—I. D., a 9 year old boy, was admitted to Babies Hospital on April 12, 1930, complaining of pain of two months' duration in the upper part of the abdomen. The pain was not related to meals. There was a history of hematemesis and tarry stools five weeks previously. A diagnosis of duodenal ulcer with hemorrhage was made on the patient's admission. While in the hospital he had tarry stools and gave 4 plus reaction to the guaiac test. He left against advice, unimproved. Follow-up observation of this patient revealed that a gastroenterostomy for duodenal ulcer with hemorrhage was performed eight years later at another hospital. In October 1944 the patient's physician reported his condition as "excellent."

CASE 3.—I. F., a 12 year old boy, was admitted to Babies Hospital on May 23, 1930 because of weakness and tarry stools. At the age of 10 years, on awakening one morning, he felt faint and vomited bright red blood; he had tarry stools that day. After this episode he frequently complained of vague abdominal pain and belching. Five days before he was admitted to the hospital he again had tarry stools and complained of weakness. The diagnosis on his admission was duodenal ulcer with hemorrhage. Roentgenographic studies revealed a duodenal ulcer with crater formation. After five weeks of conservative treatment on a Sippy regimen he was discharged much improved. On March 13, 1935 he was admitted to the Presbyterian Hospital (he was then 17 years old) with a history of seven or eight episodes of bleeding in the preceding five years, manifested by faintness, slight nausea and the passage of tarry stools. At this time it was stated, "for as long as he can remember, he has had hunger pains three to four hours after meals, which are relieved within a few minutes by any kind of food." Two months later, during his next period of hospitalization for bleeding, operation was undertaken, a duodenal ulcer being excised and a pyloroplasty performed. He made an uneventful recovery.

On Nov. 19, 1936 he was readmitted to Presbyterian Hospital for recurrent duodenal ulcer with hemorrhage and was treated conservatively, with improvement. He was hospitalized for the same condition on Aug. 30, 1939 and again on Feb. 13, 1940. He was last seen in the follow-up clinic in May 1942; at this time he was free of symptoms, having had no apparent bleeding since his last discharge from the hospital.

. Logan, G., and Walters, W.: Chronic Gastric r in Childhood Treated Surgically, Ann. Surg. 260, 1941.

. Strode, J. E.: Gastro-Jejuual Ulcer in Child, Am. J. Surg. **21**:240, 1933.

7. Shore, B. R.: Acute Ulceration of the Stomach in Children, Ann. Surg. **92**:234, 1930.

CASE 4.—A. S., a 4 month old girl, was admitted to Babies Hospital because of fever, vomiting and diarrhea for the previous twenty-four hours. The diagnosis on her admission was "intestinal intoxication of infancy." On her third day in the hospital she had five loose stools containing bright red blood. She was treated with transfusions of blood, hypodermoclyses and camphorated tincture of opium U. S. P. but died on the seventh day. Autopsy revealed the following conditions: acute duodenal ulcer with hemorrhage, acute esophagitis, acute otitis media and patent foramen ovale.

CASE 5.—R. L., an 8 month old boy, was admitted to Babies Hospital on Dec. 7, 1932, because of fever and irritability for the preceding week. The diagnosis of meningitis on his admission was confirmed by examination of the spinal fluid, from which a beta hemolytic streptococcus was isolated. The course was stormy, with spiking temperature, and the patient died in three weeks. Treatment consisted in myringotomy and blood transfusions. The conditions observed at necropsy were: meningitis (beta hemolytic streptococcus), hydronephrosis, hemosiderosis and duodenal ulcer.

CASE 6.—A. B., a 10 year old girl, was admitted to Babies Hospital on Feb. 19, 1934, with a history of pain in the right upper abdominal quadrant for the previous two years. The pain bore no relation to meals but was relieved by sodium bicarbonate. About one year before her admission she had an episode of hematemesis, which was followed by tarry stools for a few days. The diagnosis on her admission was peptic ulcer. Roentgenographic examination of the gastrointestinal tract showed a duodenal ulcer niche. Examination of the stool showed the presence of blood. Gastric analysis revealed a total acidity of 79 degrees and free hydrochloride acid of 25 degrees before administration of histamine. Twenty minutes after administration of histamine the total acidity was 55 degrees and the free hydrochloric acid 45 degrees. Forty minutes after administration of histamine the total acidity was 59 degrees and the free hydrochloric acid 54 degrees. The patient was placed on a medical regimen designed for patients with ulcer and was discharged in nine days. In April and, again, in September 1934 roentgenographic examination revealed no evidence of ulcer. On subsequent visits in January, April and October 1935 she was still free from symptoms.

CASE 7.—S. G., a 12 year old boy, was admitted to Babies Hospital on Dec. 24, 1935, with a history of epigastric pain and vomiting of two months' duration. The pain had no relation to meals. The diagnosis on his admission was "abdominal pain of unknown origin." Roentgenographic examination showed nothing abnormal except for a low position of the transverse colon. The patient was discharged in two days, no diagnosis having been made. He was readmitted in five months with recurrence of the epigastric pain; at this time he also had tarry stools and weakness. The diagnosis on this admission was abdominal pain and melena. Roentgenologic studies again revealed no abnormalities. Laparotomy was undertaken for a possible Meckel diverticulum. Appendectomy was performed, and a duodenal ulcer was noted. A diverticulum was not found. He was subsequently transferred because of his age to the adult service of the Presbyterian Hospital and was discharged after a diet appropriate for a person with ulcer had been prescribed.

Two months later he was readmitted for duodenal ulcer with hemorrhage. Within seven years there were five additional admissions to the Presbyterian Hospital for the same condition. During his last stay in the hospital, beginning Jan. 31, 1944, when he was [illegible] partial gastrectomy was performed. He was last [illegible] in the follow-up clinic, in June 1944; at that time was free from symptoms.

CASE 8.—H. E., a 3 year old boy, was admitted to Babies Hospital because of high fever for four [illegible]. The diagnosis on his admission was nephrosis and [illegible] tonitis. The patient died in three weeks. The [illegible] tions observed at necropsy were: acute peritonitis, [illegible] gional infected fistula of the umbilicus, lobar pneumonia (type VI), nephrosis, acute ulcer of the duodenum with hemorrhage and acute ulcer of the esophagus.

CASE 9.—K. G., a 3 month old girl, was admitted to Babies Hospital on Nov. 14, 1942, with congenital hydrocephalus. Bilateral cauterization of the choroid plexus was performed. This procedure was followed by the appearance of decerebrate rigidity, which neurologists attributed to injury of the basal ganglia. The patient died on her sixteenth day in the hospital. There was a perforation of the stomach due to [illegible] which was interpreted as of neurogenic origin.

DETAILED REPORT OF A CASE

CASE 10.—On March 27, 1944 A. S., a 12 year old undernourished, highly emotional Puerto Rican girl was admitted to the medical service of Babies Hospital complaining of abdominal pain and vomiting.

History.—He had been well up to the age of 8 years when, without any known precipitating factor, he began to complain of generalized abdominal pain, which gradually increased in severity. The pain was nonradiating and nonlocalized and had no particular relation to the taking of food. The attacks were intermittent, lasting for thirty to sixty minutes over a period of two or three weeks. Between these attacks he was free from symptoms. He had lost weight, the amount being unknown. One week previous to his admission he was awakened from sleep by epigastric pain and fullness which were followed by nausea and vomiting. The vomitus consisted of particles of food and was described as having a foul odor and as not containing any blood. After this episode the patient complained of marked eructation, epigastric pain and fulness, which were relieved by vomiting about one hour after meals. These manifestations occurred two or three times daily. Because of the relief afforded, vomiting was often self-induced.

The remainder of the personal history and the family history were noncontributory.

Examination.—Physical examination did not reveal anything abnormal.

The urine, the blood count and the chemical constituents of the blood were all normal. Examination of stools failed to reveal the presence of blood. A gastric analysis, on April 8, gave the following data:

	Approximate Amounts, Cc.	Free HCl, Degrees	Total Acidity, Degrees
Fasting	48.0	11.5	37.5
20 minutes after histamine	72.5	9.7	32.5
40 minutes after histamine	80.0	22.0	55.0
60 minutes after histamine	30.0	25.5	57.0

Roentgenographic visualization of the gastrointestinal tract on March 21 showed ". . . the stomach outlet to be obstructed. After seven hours it had a 50 per cent retention. The duodenal cap was small but not deformed. No ulcer crater was visualized." On March 31 a second examination showed a residue of food

omach after fifteen hours. Studies of the duo- suggested an ulcer in its second portion. Roent- gists also mentioned the possibility of a congenital narrowing the duodenum and causing obstruction.

erative Procedure.—A laparotomy was performed pril 28, through an upper right rectus incision, with atient under ether anesthesia induced by the open method. On the anterior surface of the lower end e first portion of the duodenum a well defined s" ulcer, with considerable serosal scarring and ening, was encountered. In view of the low acid , the obstruction and the age of the patient a rior gastroenterostomy was performed instead of total resection.

urse.—Convalescence was entirely uneventful, and patient was discharged on the fourteenth post- tive day after a diet appropriate for a convalescent an ulcer had been prescribed.

llow-up of this patient revealed that on May 26, he experienced some pain after eating a ham vich, that on June 23 he was free from symptoms hat on August 8 he was still free from symptoms was gaining weight. He was last seen on Decem- l, eight months after operation. He was free from toms and weighed 100 pounds (45.4 Kg.), a gain pounds (6.8 Kg.), since his operation.

SUMMARY AND CONCLUSIONS

Although gastric and duodenal ulceration is rare in infancy and childhood, the diagnosis would probably be made more frequently if it were realized that the condition does occur in this age group.

We have found hemorrhage the most common complication. It occurred in 6 of the 10 cases reported. Perforation and pyloric stenosis were also observed. Malignant change has not been encountered.

The indications for surgical intervention in the treatment of gastric and duodenal ulcers in infancy and childhood are much the same as those in adults, namely, perforation, obstruction, recurrent hemorrhage and intractable pain. One apparently need not be concerned with carcinoma in treating children of the age group under consideration.

862 Park Avenue.

Abstracts from Current Literature

Anatomy and Physiology

A Simple Technic for Studying the Periodicity and Survival of the Respiratory Center. W. A. Selle, Proc. Soc. Exper. Biol. & Med. **54**:291 (Dec.) 1943.

In a previous study with isolated, unperfused heads of white rats the author found that after cessation of the flow of blood gasping continued for some time. The duration and the total number of gasps were inversely proportional to the age up to the period of weaning. Two types of gasping movements were observed: the initial, or aerobic, phase (I) consisted of 6 to 12 gasps and lasted from fifteen to forty seconds; the second, or anaerobic, phase (II) did not begin before twenty to fifty seconds after the cessation of the first and lasted from twenty-five to thirty minutes in newborn rats to two to three minutes in 21 day old rats. Rats 5 or 6 weeks of age or older did not have the second phase. Similar observations were made with rabbits, cats and dogs (Selle: *Proc. Soc. Exper. Biol. & Med.* **48**:417, 1941).

In the present study, with controlled rats 12 to 15 days of age the following observations were made: The survival of the respiratory center varied with the amount of dextrose in the blood; the gasps of type I were inhibited by cyanide; the gasps of type II were inhibited by iodoacetic acid; survival was shortened by administration of dinitrophenol and thyroxin; gasping was depressed by hypnotics and general anesthetics and stimulated by alcohol, morphine and a preparation of chloral hydrate and dextrose (chloralose); metrazol counteracted barbiturate depression. Rapid cooling of the isolated heads by immersing them in water or by spraying them with ethyl chloride U. S. P. resulted in suspension of gasping, which was revived when they were again warmed. In 1 to 4 day old rats activity was resumed after more than fifty minutes of cooling.

Hansen, Galveston, Texas.

Biochemistry; Bacteriology, and Pathology

The Behavior of Virulent and Avirulent P. Pestis in Normal and Immune Experimental Animals. E. Jawetz and K. F. Meyer, J. Infect. Dis. **74**:1 (Jan.-Feb.) 1944.

After the subcutaneous or intracutaneous administration of virulent Pasteurella pestis into mice, guinea pigs or rats, the organisms invade the bodies of these experimental animals in the same sequence of lymphatic and hematogenic spread demonstrated for many other bacteria. With the use of a sensitive culture technic, it is possible to demonstrate that the distribution of similarly injected avirulent P. pestis differs quantitatively but not qualitatively from that observed with virulent bacilli.

The immunogenic activity of a number of avirulent strains of plague was found to differ significantly for different species of animals, for example, guinea pig and mouse. Since, however, these strains of plague are distributed equally and persist for the same length of time in the different host animals, it appears that immunogenic activity depends not on the invasiveness or the survival of a plague organism but rather o antigenic makeup. The avirulent strains of P. p tested in these experiments survive for a longer pe in guinea pigs than in mice.

Actively immunized guinea pigs and mice inocul with a large dose of virulent plague bacilli sho rapid initial distribution of organisms throughout body, similar to that observed in normal animals. A a few days, however, the virulent bacilli disappear f the immune tissues in the following sequence: bl stream, liver and spleen, regional lymph nodes local lesion. In normal susceptible host animals viru bacilli multiply steadily until death occurs.

Avirulent plague bacilli introduced subcutaneously large, toxic numbers cause the death of a high portion of the inoculated mice. The cumulative m tality curve observed in such an experiment clo resembles that found after administration of a m virulent dose. The reasons for this similarity and possible significance are discussed.

Intracerebrally administered avirulent plague ba survive for a significantly longer period in the br of mice than avirulent bacilli administered by any o route. This observation may help in the understand of chronic latent plague meningitis and encephal cases of which have been observed in California.

The gross and microscopic pathologic changes brou about by the injection of avirulent live plague bacilli of extract derived from them are described and significance of the observations is discussed.

Toomey, Clevelan

Metabolism; Infant Feeding Milk and Other Foods

The Early Recognition of War Nutrition De ciency in Children. I. Newton Kugelmass, M J. Digest. Dis. **11**:83 (March) 1944.

A review of the causes of deficiency diseases in c dren is given. The daily nutrient requirements of p tein, calcium, iron and vitamins A, B_1, C. G, PP and are given for the periods of infancy, childhood, pube and adolescence. Early physical, cutaneous, digesti respiratory, sensory, mental and nervous manifestati of nutritional deficiency are cited.

This long article lends itself poorly to abstraction s should be read in its entirety.

Morrison, Savannah, Ga

Observations on a Diet Deficient in Both Met onine and Cystine in Man. Anthony A. A banese, L. Emmett Holt Jr., Joseph E. Bru back Jr., Jane Frankston and Virginia In Bull. Johns Hopkins Hosp. **74**:308 (May) 1944.

In 2 healthy male subjects given a diet deficient both sulfur amino acids (methionine and cystine) ne tive nitrogen balances developed. Nitrogen equilibri was restored by the addition of a supplement of met onine alone, a result which suggests that methion is capable of furnishing the entire requirement of m for sulfur amino acids.

Authors' Summary

SFERS OF INTRACELLULAR POTASSIUM IN EXPERI-NTAL DEHYDRATION. J. RUSSELL ELKINGTON and LEXANDER W. WINKLER, J. Clin. Investigation **23**: (Jan.) 1944.

s of intracellular potassium in excess of that asso- with the catabolism of protein is a general se to depletion of water from any cause. Loss of ellular water accompanies this loss of excess po- m, with the result that the loss of extracellular is minimized. Renal activity is essential to effect oss of potassium.

ese conclusions are based on extensive data, cited paper, on mineral, nitrogen and water balances in subjected to various experimental procedures ed to cause rapid depletion of water in the body.

GUEST, Cincinnati.

MPLE LIVER PREPARATION ORALLY EFFECTIVE IN E TREATMENT OF HUMAN AMYLOID DISEASE. MES LOUKIDES, HAROLD G. GRAYZEL and MENDEL COBI, J. Lab. & Clin. Md. **28**:1321 (Aug.) 1943.

e authors describe the preparation of a "liver cock- a mixture of fresh liver and fruit juice which has found effective in the treatment of amyloid disease.

WILKINSON, Galveston, Texas.

HER STUDIES ON THE COMPARATIVE VALUE OF UTTER FAT, VEGETABLE OILS AND OLEOMARGARINES. . K. BOUTWELL, R. P. GEYER, C. A. ELVEHJEM d E. B. HART, J. Nutrition **26**:601 (Dec.) 1943.

K. Boutwell and others present data showing that nterrelationship exists between the kind of carbo- ate and the kind of fat that are combined. With se as the sole carbohydrate, the vegetable oils are itely inferior to butter fat and lard. With a mix- of carbohydrates submitted for lactose, approxi- ly equal growth was attained by rats fed either table or animal fats, and growth was superior to attained by rats which were fed lactose.

FREDEEN, Kansas City, Mo.

PROTEIN NUTRITIONAL VALUE OF SOYBEAN, PEA-UT AND COTTONSEED FLOURS AND THEIR VALUE S SUPPLEMENTS TO WHEAT FLOUR. D. BREESE ONES and J. P. DIVINE, J. Nutrition **28**:41 (July) 944.

ybean, peanut and cottonseed flours contain proteins igh nutritive value and offer an excellent means of lying dietary protein to extend and partially replace ein foods of animal origin. D. Breese Jones and . Divine compared the growth-promoting values of e proteins by the rat growth method. These plant eins greatly enhanced the nutritive value of the pro- of wheat flour, even when as little as 5 per cent added to the wheat flour. The mixtures contained n 16 to 19 per cent more protein than wheat flour e and were definitely superior to it in their growth- noting value.

FREDEEN, Kansas City, Mo.

SCRIBED DIETS FOR NORMAL CHILDREN. JULIAN D. OYD, J. Pediat. **24**:616 (June) 1944.

any benefits are to be expected through the better- t of nutrition in childhood. The goal should be provision of the diet best fitted to meet the child's test needs. An optimum state of nutrition cannot xpected if the child's practices in eating are casual unsupervised. When the usual child's dietary is analyzed various deficiencies become apparent. Information is available as to the qualitative and the quantitative nutritional needs of the child and as to the manner of meeting these needs through the use of simple foodstuffs. A plan of dietary is offered, which meets completely the standards set up by the Food and Nutrition Board of the National Research Council and which is made up entirely of commonly available foodstuffs. Each food is discussed as to the qualitative and the quantitative contribution it makes toward meeting the day's quota of the various specific nutritional factors. The need for consideration of the supply of energy is stressed and emphasis offered that a diet may meet all needs for protein, minerals and vitamins and yet be grossly deficient in calories. AUTHOR'S SUMMARY.

EFFECT OF PARENTERALLY ADMINISTERED CITRATE ON THE RENAL EXCRETION OF CALCIUM. GEORGE GOMORI and ELSIE GULYAS, Proc. Soc. Exper. Biol. & Med. **56**:226 (June) 1944.

Sodium citrate in 4 per cent solution, 8 to 30 cc. per kilogram of body weight, was injected parenterally into 7 adult dogs, 4 puppies and 6 rats. A study of the calcium and phosphorus in the blood and in the urine of the dogs gave the following results: There was a marked increase in the level of urinary calcium after about one hour; it returned to normal in five hours. A tendency to the late excretion of urinary phosphorus was noted. No changes occurred in levels of calcium and of phosphorus in the serum. From postmortem examination of 1 puppy which died during the experiment and from biopsies of the other animals it was found that typical changes in the bones occurred. The bone marrow showed hyperemia and edema, and in addition there was transformation of osteoblasts into spindle cells, increase in the number of osteoclasts, fragmentation of bone trabeculae and areas of early fibrosis. Rats given the same treatment also presented this picture, which was extremely similar to that seen after toxic doses of parathyroid extract.

HANSEN, Galveston, Texas.

THE PLATANO, MUSA PARADISIACA OF LINNAEUS, IN INFANT FEEDING. RAFAEL DE LA PORTILLA, Lavastida Bol. Soc. cubana de pediat. **15**:153 (April) 1943.

The author refers to a kind of banana, common in tropical countries, eaten green or ripe but only after cooking or other preparation. It is fed to infants in the form of a powder, which can be made in the home by crushing and sieving the banana or obtained in the form of a commercially dehydrated product, Bananina. The chemical composition of the fruit is given, with emphasis on its high content of carbohydrate and its high caloric value, one tablespoonful of the powder containing 39.69 calories. It is rich in iron, magnesium and sodium but poor in calcium. It is also especially rich in vitamins A and B, and it is easily digested because of its enzymes and its low residue.

From his own observations the author concludes that the powdered banana is useful as food for normal infants as young as 3 months because it supplies calories, minerals and vitamins and can be used with whole or with evaporated milk. It is easily digested by the undernourished infant and is effective in the treatment of intestinal disturbance when diarrhea predominates, as it apparently has bacteriostatic properties. It is also used in the treatment of hypochromic anemia and of avitaminosis.

KEITH, Rochester, Minn.

Vitamins; Avitaminoses

VITAMIN CONTENT OF VARIETY MEATS. J. M. McINTIRE, B. S. SCHWEIGERT, E. J. HERBST and C. A. ELVEHJEM, J. Nutrition **28**:35 (July) 1944.

J. M. McIntire and others determined the amounts of thiamine, riboflavin and nicotinic acid in variety meats. Prepared meats were found to be a good source of these vitamins. They contain about the same amounts as fresh muscle meats. See the tables in the article.

FREDEEN, Kansas City, Mo.

THE UTILIZATION OF THIAMINE IN THE HUMAN SUBJECT: THE EFFECT OF HIGH INTAKE OF CARBOHYDRATE OR OF FAT. JOHN G. REINHOLD, JOSEPH T. L. NICHOLSON and K. O'SHEA ELSOM, J. Nutrition **28**:51 (July) 1944.

J. G. Reinhold and others found that the decrease in the excretion of thiamine observed when diets high in carbohydrate were fed indicates that the proportion of carbohydrate in the diet is an important factor in determining the daily requirement of thiamine, in view of the dependence of excretion of thiamine in urine on the utilization of thiamine when intake and other factors are kept constant.

FREDEEN, Kansas City, Mo.

THE PRODUCTION OF VITAMINS IN GERMINATED PEAS, SOYBEANS AND OTHER BEANS. CYRUS E. FRENCH, GEORGE H. BERRYMAN, JOHN T. GOORLEY, HAROLD A. HARPER, DAVID M. HARKNESS and EDWARD J. THACKER, J. Nutrition **28**:63 (July) 1944.

Cyrus E. French and others found that germination caused a marked increase in ascorbic acid, a fair increase in riboflavin and nicotinic acid and no increase in thiamine. This was in addition to the vitamin content inherent in the nongerminated legume.

FREDEEN, Kansas City, Mo.

THE HEALING OF RENAL RICKETS. WILFRID SHELDON, Arch. Dis. Childhood **18**:194 (Dec.) 1943.

The case of a boy aged 4 years with chronic nephritis and renal rickets is recorded. The bones of this patient underwent complete healing under treatment with alkalis, calcium and large amounts of vitamin D. The recovery is regarded as not fortuitous, but it is not possible to decide whether it was due to one therapeutic measure alone or to the combined therapy.

KELLY, Milwaukee.

Hygiene; Growth and Nutrition; Public Health

UNITS OF LOCAL HEALTH SERVICE FOR ALL THE STATES. Committee on Local Health Units, Am. J. Pub. Health **33**:404 (April) 1943.

This is a report of progress of the subcommittee on local health units of the committee on administrative practice of the American Public Health Association. This committee was created to review the existing full time local health services in each state and to prepare information in a practical form for use in promoting the extension of such services to all the people of the United States.

There are over 40,000,000 people in more than half of the country's 3,070 counties for whom no full time health services are provided (as of July 1942). Using criteria of population, geography, economic status and availability of physicians and of hospital beds, the committee proposes the creation of 1,127 units of local he jurisdiction, to be staffed by full time qualified sonnel. Of these, 303 would include a single co 677 would include two to four counties and 147 w include more than four counties each.

PLATT, Rochester, N.

A NEGRO DEMONSTRATION CENTER FOR MATERNAL NEWBORN CARE IN ALABAMA. T. M. BOULW ELIZABETH LAFORGE and R. C. STEWART, South. **36**:784 (Dec.) 1943.

This is a detailed report of a successful effort to prove maternal and neonatal care among the N population of a section of a southern industrial The center not only renders service to patients but training center for Negro physicians and nurses.

LOGAN, Rochester, Min

Prematurity and Congenital Deformities

SPINA BIFIDA AND ITS SKULL DEFECTS. J. B HARTLEY and C. W. F. BURNETT, Arch. Dis. C hood **18**:173 (Dec.) 1943.

Craniolacunia consists of an irregular ossificatio the inner surfaces of the bones of the vault of the f skull, whereby thick bony bars or ridges are produ which branch and unite in such a way as to for reticulated pattern of circles and ellipses. Between bars exist thinned areas of bone, which form the lacu from which the condition is named. In craniofenes the defective ossification is carried a stage further, areas marked by the absence of bone formation oc between the bars, producing a pattern of fenestrat

The rate of incidence of these developmental anom is nearly 1 per cent of all births.

Their frequent and little known association with sp bifida is emphasized.

It is suggested that the laws governing the inherita of spina bifida are most likely to be discovered b study of the relatively common developmental de namely, craniolacunia and craniofenestria. The mot of infants with these defects should likewise be stud both immediately and during any future pregnancy.

KELLY, Milwaukee.

A CASE SHOWING PARTIAL DEFICIENT FUSION O MAXILLARY PROCESS WITH LATERAL NASAL PROC ON ONE SIDE. G. E. DODDS, Brit. J. Ophth. **27**: (Sept.) 1943.

A Negro girl aged 3 months presented the congen abnormalities to be described. The outstanding de was distortion of the right nostril, with a hiatus show the interior of the nose; the cleft extended upward within 1 cm. of the inner canthus, which had a w obtuse angle. There were small notches, or colobom in both upper lids and a notch in the right lower l There was no caruncle, and the presence of a p semilunaris was doubtful. On the inner side of medial canthus of the left eye there was a verti hole, in appearance like the opening of a sinus. cleft, or fissure, extended from the lower and late margin of the right nasal bone to a point where n mally the nostril joins the lip. The latter was merel cleft in the cartilaginous part of the nose. The e were normal in every respect.

A sketch accompanies the article.

ZENTMAYER. [ARCH. OPHTH.]

Newborn

INFLUENCE OF NUTRITION DURING PREGNANCY UPON THE CONDITION OF THE INFANT AT BIRTH. BERTHA S. BURKE, VIRGINIA A. BEAL, SAMUEL B. KIRKWOOD and HAROLD C. STUART, J. Nutrition **26**: 569 (Dec.) 1943.

Bertha S. Burke and others have shown a statistically significant relationship between the diet of the mother during pregnancy and the condition of her infant at birth and during the neonatal period. A good or excellent diet produced an infant in good or excellent physical condition, and a poor antepartum diet resulted in an infant in poor physical condition.

FREDEEN, Kansas City, Mo.

COMPARISON OF NEWBORN INFANTS WITH ERYTHROBLASTOSIS FETALIS AND THOSE BORN TO DIABETIC MOTHERS. H. C. MILLER, R. D. JOHNSON and S. H. DURLACHER, J. Pediat. **24**:603 (June) 1944.

Marked similarities and differences exist during the first few days of life in infants born to diabetic mothers and infants with erythroblastosis fetalis. The similarities include increased normoblastemia, excessive hematopoiesis in the liver and other viscera, temporary cardiac hypertrophy, adrenal hyperplasia and extensive petechial and gross hemorrhages in all tissues. The two groups of infants differ in that anemia and jaundice are rarely encountered in infants born to diabetic mothers. The distribution of the Rh factor in the blood of diabetic mothers is like that in the population at large, whereas all of the mothers studied whose infants had erythroblastosis fetalis were Rh negative and their infants Rh positive.

It is concluded that the inclusion of infants born to diabetic and "prediabetic" mothers in previous studies of the syndrome "erythroblastosis fetalis" accounts for the fact that 10 per cent of the mothers in those studies were Rh positive. The possible relationship of the Rh factor to the observations in infants born to diabetic mothers and in infants with erythroblastosis fetalis is discussed.

AUTHORS' SUMMARY.

AUTONOMIC NERVOUS SYSTEM IMBALANCE OF THE WHOLE GASTROINTESTINAL TRACT IN THE NEWBORN. GERARD N. KROST, J. Pediat. **24**:635 (June) 1944.

Two infants were observed with marked symptoms of gastrointestinal disease due to faulty innervation of the gastrointestinal tract by the autonomic nervous system. The symptoms were not unlike those accompanying postoperative paralytic ileus. Both infants died from terminal adhesive peritonitis and from malnutrition. Both were subjected to exploratory laparotomy. The first was observed when 20 days old, the second when only 13 days old. In neither was any obstruction found.

The symptoms in the first patient began with vomiting on the third day of life. A barium gastrointestinal roentgen examination was done on the same day. There was marked delay in the emptying of the stomach. Barium was present in the stomach up to nine hours. Barium reached the colon in twenty-four hours. Evidence of distention of the bowel was reported. Vomiting was checked for about two weeks by thick feedings. Then vomiting recurred, and putrefactive diarrhea developed when the infant was 14 days old. The abdomen became much distended, and loops of bowel were visible across it. Exploratory laparotomy was done when the infant was 20 days old. Greatly distended loops of bowel, with segmented spasm, were found, but no obstruction. The patient followed a steady downhill course, with persistent diarrhea of putrefactive odor. Enemas revealed a stasis of the large bowel, though diarrhea existed. Atropine failed to decrease the vomiting. Neostigmine administered by mouth, 2 mg. every eight hours, had no effect on the distention. Death occurred at the age of 2 months and 24 days. Postmortem examination revealed marked distention of the entire bowel and chronic adhesive peritonitis.

The second patient was seen when 13 days old. He was a large baby at birth, and delivery was difficult. When he was 3 days old marked distention of the abdomen occurred. This condition was relieved by enemas. When the patient was seen at home, his symptoms were moderate distention of the abdomen, dehydration and regurgitation of food if he was given more than 2 to 12.5 ounces (71 Gm.) and putrefactive diarrhea. He was sent to the hospital when 15 days old. Gastrointestinal roentgen examination after a barium meal gave these findings: The pylorus opened after a long delay, approximately one hour. Opaque material could be expressed through the pylorus by manipulation. A small residue remained in the stomach at the end of six hours. The colon was distended. A few loops of small bowel were distended with gas. Supportive treatment was given, consisting of subcutaneous administration of fluids and transfusions of plasma. When the patient was 21 days old an exploratory operation was done. Aside from a marked Jackson's membrane, which was severed, and moderate distention of the whole bowel, there were no findings. There was no improvement. Atropine did not affect the regurgitation. Administration of neostigmine bromide, 1:4,000 solution (0.5 cc. every twelve hours), was started on the twelfth day in the hospital. This treatment seemed to relieve the distention markedly. Diarrhea with a putrefactive odor continued. The existence of stasis was evident from the large quantities of stool brought by enema and from the presence of fecal masses which could be palpated through the abdominal wall, even though diarrhea was present. Some improvement was shown for about two weeks after administration of neostigmine was started, but projectile vomiting recurred. Universal edema developed. Transfusions and subcutaneous fluids prescribed as supportive measures were of no avail. Death occurred when the patient was 2 months and 9 days old. The changes observed at autopsy were those of a chronic adhesive peritonitis, marked distention and thinning of the small bowel and distention of the colon. The medullary portion of the adrenals was congested and hemorrhagic; the cortical portion was quite edematous. The liver was fatty and parenchymatous, with marked degeneration. The other organs were normal.

Each of these infants displayed symptoms of generalized atony of the gastrointestinal tract, which was probably caused by imbalance of the autonomic nervous system. Each had putrefactive diarrhea which might be explained by faulty vagal stimulation of the pancreas and by a decrease in the secretion of trypsin. The parasympathetic stimulating preparation neostigmine helped the second patient for a short time. In neither patient was there any disease of the pancreas. In neither was there a true picture of congenital megacolon. Each was subjected to exploratory laparotomy on the mistaken hypothesis that a partial obstruction was present.

AUTHOR'S SUMMARY.

NUCLEATED ERYTHROCYTES IN NEWLY-BORN INFANTS IN RELATION TO MATERNAL RH COMPATIBILITY. ALBERT E. CASEY and SARA H. CROWSON, Proc. Soc. Exper. Biol. & Med. **54**:320 (Dec.) 1943.

The number of nucleated erythrocytes per 10,000 red cells was counted in 116 newborn infants. Both the mother and the child had Rh-positive blood in 81 instances and Rh-negative blood in 11 instances. In 16 cases the mother's blood was Rh-negative and the infant's Rh-positive, and in 8 the mother's blood was Rh-positive and the infant's Rh-negative. Of the 92 infants who had the same Rh reaction as the mother, 6 died within a few days, and in 4 of these the count of nucleated erythrocytes was higher than normal. Among the 24 infants with a different Rh reaction from that of the mother, 7 had 2 or more nucleated cells per 10,000 red cells. The authors recommend that a count of nucleated erythrocytes be made for every newborn infant, and they state that 2 or more nucleated red blood cells per 10,000 are to be considered abnormal. Regardless of Rh compatibility, the mortality among infants with 5 or more nucleated cells per 10,000 red cells was 67 per cent in this series of cases. The possibility of ill effects when the mother's blood is Rh-positive and the infant's Rh-negative is suggested by the data of the authors. HANSEN, Galveston, Texas.

TRANSECTION OF THE SPINAL CORD DUE TO INJURY AT BIRTH. MARGARET HORAN, Arch. Dis. Childhood **18**:188 (Dec.) 1943.

A case is described of complete transection of the spinal cord in an infant.

Reasons are given for considering that the condition was traumatic in origin and was produced by traction on the body of the child during an unusual breech delivery.

It is suggested that obscure diseases of the lower limbs may be related to injuries of the spinal cord at birth. KELLY, Milwaukee.

Acute Contagious Diseases

COMPARATIVE SPEED OF RESPONSE OF PREVIOUSLY IMMUNIZED AND NONIMMUNIZED CHILDREN TO FLUID AND ALUM-PRECIPITATED · DIPHTHERIA TOXOID. VLADIMER K. VOLK, WILLIAM EDWARD BUNNEY and JOHN T. TRIPP, Am. J. Pub. Health **33**:475 (May) 1943.

Immunity was measured by determining the antitoxin content of the blood of children just before the injection for reimmunization and at five and ten days respectively after the injection. Included in the study were 112 children previously immunized. Most of the children were from 6 to 9 years of age at the time of the primary immunization. They were reimmunized approximately four years later.

There were 98 children who had previously received a complete immunizing treatment (at least one dose of alum-precipitated toxoid or at least two doses of fluid toxoid). Of these 57 had less than 0.001 unit of antitoxin per cubic centimeter of serum. Ten days after the injection of 0.5 cc. of either fluid or alum-precipitated toxoid, 96 per cent had more than 0.001 unit per cubic centimeter of serum and 82 per cent had 0.1 unit or more. Of the 41 children who had more than 0.001 unit per cubic centimeter of serum before the restimulating injection, 95 per cent had 0.1 unit or more per cubic centimeter ten days after the injection. The suggests that fluid toxoid is just as effective as precipitated toxoid in eliciting a quick response persons previously immunized. As a control, 87 dren with no previous history of immunization with levels of antitoxin of less than 0.001 unit per centimeter of serum received similar injections. In 25 per cent did 0.001 unit or more of antitoxin de within ten days.

Although the numbers studied were small, it ap that after primary immunization with one injecti alum-precipitated toxoid or two injections of toxoid another injection of either type of toxoid give a rapid response. Such a response will occ any time during at least five years after the pri immunization.

When a community faces an epidemic of diphth the authors recommend the administration of a re ulating dose of toxoid to all children previ immunized. PLATT, Rochester, N.

A SMALL OUTBREAK OF SMALLPOX IN DE FRANKLIN H. TOP and LAURA E. PECK, Am Pub. Health **33**:490 (May) 1943.

This paper reviews morbidity and mortality rate smallpox in the United States and describes a s outbreak of smallpox in Detroit in 1940.

During the twenty year period ending in 1939 70 cases of smallpox, with 5,337 deaths, were report the United States. The case fatality rate of less 1 per cent was due to the fact that in most of t cases the disease was of the alastrim type. During past forty years there has been a progressive decr in the number of cases of severe smallpox and a comitant decrease in the number of deaths. How severe smallpox is still present and may reappea epidemic form at any time. For the ten year pe ending in 1941 the morbidity rates of seven st which forbid compulsory vaccination were thir times as great as those in eleven states which re vaccination as a prerequisite to attendance at scho

In 1940 an epidemic of 18 cases of smallpox occu in Detroit. The early cases were diagnosed as chic pox. None of the patients had ever been successf vaccinated. The incubation periods ranged from to twenty-five days, but in 14 cases the range between fourteen and seventeen days.

PLATT, Rochester, N.

COMPARISON OF THE NASOPHARYNGEAL SWAB AND COUGH PLATE IN THE DIAGNOSIS OF WHOO COUGH AND HEMOPHILUS PERTUSSIS CARR JOHN J. MILLER JR., CHARLES W. LEACH, T. SAITO and J. B. HUMBER, Am. J. Pub. Health 839 (July) 1943.

One or more duplicate tests with nasopharyn swabs and cough plates were made on 214 children had had a diagnosis of pertussis made on the basi typical paroxysms, with or without hyperlymphocyt The cultures were obtained by many different phy cians and nurses. A total of 342 comparative cult were examined. The combination of the two meth gave more positive results than either method al For every age group and every week of the dis the swab gave a higher percentage of positive re than did the cough plate. During the first week the disease 80 per cent of the patients showed pos

ions by one method or the other. In the fifth only 32 per cent gave positive reactions.

elve persons, including 8 adults who had been in act with children with bacteriologically proved per-s, were also examined. Cough plates from 2 chil- and from 1 adult showed positive reactions. One ese persons began to cough three days later; the 2 remained well. The duration of the healthy er state was not determined.

e authors conclude that the nasopharyngeal swab ic provides a relatively simple means of detecting instances, atypical instances and carriers.

PLATT, Rochester, N. Y.

M AMYLASE IN MUMPS. I. L. APPLEBAUM, Ann. nt. Med. **21**:35 (July) 1944.

rum amylase was found in amounts greater than al during the active stage of mumps in a series patients. The author attaches importance to this rvation for the diagnosis of mumps, especially in ical cases.

READING, Galveston, Tex.

NTILE PARALYSIS. LENOX D. BAKER and HOWARD CHAUBEL, Arch. Surg. **47**:384 (Oct.) 1943.

an excellent review of this subject the recent ature on the various phases of infantile paralysis iscussed. Of particular interest is a summary of published papers on tonsillectomy and infantile lysis. Of greatest significance in these reviews is statement by Aycock that the absence of tonsils not predispose to the paralysis of poliomyelitis rather is a determinant as between the spinal and bulbar type of the disease.

KAISER, Rochester, N. Y.

GENITAL CHICKENPOX WITH DISSEMINATED VIS-ERAL LESIONS. ELLA H. OPPENHEIMER, Bull. Johns Hopkins Hosp. **74**:240 (May) 1944.

case of congenital chickenpox in which the intra-ine transmission of the virus occurred at least five before birth is described. Autopsy at 11 days ed disseminated necrosis with epithelial intranu-inclusion bodies in the skin, the intestines and ous organs.

LYTTLE, Los Angeles.

IVE AND PASSIVE IMMUNITY IN EXPERIMENTAL HEMOPHILUS PERTUSSIS INFECTION IN MICE. JOHN A. TOOMEY, NORMAN LEWIS, EARL AVERILL, WAL-TER DRURY and WILLIAM S. TAKACS, J. Lab. & Clin. Med. **29**:21 (Jan.) 1944.

n the experiments described freshly isolated phase A ase I of Leslie and Gardner) Hemophilus pertussis anisms gave but slight protection to white mice n an attempt was made to immunize the animals ively with those organisms. The mortality rate was per cent, as compared with 99 per cent for the trols.

Only slight passive protection was given by con-escent human serum. One commercial horse anti-im gave a slight degree of protection (71.8 per cent rtality). Antipertussis rabbit serum gave better pro-ion, with a mortality of 44.2 per cent, and the best tection was obtained by the use of human hyper-mune serum (30.6 per cent mortality).

WILKINSON, Galveston, Tex.

PURPURIC LESIONS IN MENINGOCOCCIC INFECTIONS. WILLIAM G. BERNHARD and ARTHUR C. JORDAN, J. Lab. & Clin. Med. **29**:273 (March) 1944.

Smears and cultures from purpuric lesions in patients with meningococcemia offer a rapid means of diagnosis. Of 40 patients with meningococcic infection, meningococci were found in smears from the purpuric lesions of 27 (67 per cent). The organisms from 35 patients (87.5 per cent) were isolated on culture.

WILKINSON, Galveston, Tex.

WHOOPING COUGH MORTALITY. MAX J. FOX and ELIZABETH M. KNOTT, J. Pediat. **24**:671 (June) 1944.

In a recent review of the statistics for whooping cough among patients admitted to the Isolation Hospital of the Milwaukee Health Department it was discovered that 31 deaths had occurred among the 362 children admitted for this disease during the ten year period from Jan. 1, 1933 to June 30, 1943. During the same period 29,845 cases of whooping cough, with 75 fatalities, were reported to the city health office. Both in the city as a whole and in the cases isolated at the hospital for contagious diseases the severity of the disease was greatest in younger children, and in each group over 70 per cent of the deaths occurred in infants less than 1 year of age. Thus any efforts toward reducing mortality due to whooping cough must be directed toward protecting small infants.

A review of recent literature regarding immunization to whooping cough brings out the fact that the opsonocytophagic reaction is adequate when injections of Hemophilus pertussis vaccine are given to children as young as 3 months af age. Also the agglutinating reaction produces a satisfactory plateau of antibody titer in from two to four months when three divided doses of the vaccine are given at intervals of from seven to ten days. Prolonging the interval between injections has no appreciable effect on the height or the persistence of the agglutinins, but revaccination one year or more later leads to increases in titer, which can then be maintained at high levels for several years.

Because of the seriously high level of mortality among infants contracting whooping cough, the following program of immunization is suggested:

1. Begin vaccination when infants are 3 months of age.
2. Give 80,000,000,000 organisms in three divided doses at intervals of from seven to ten days.
3. Administer a stimulating dose of pertussis vaccine concurrently with diphtheria immunization toward the end of the first year.

FOX, Milwaukee.

FULMINATING MENINGOCOCCEMIA (THE WATERHOUSE-FRIDERICHSEN SYNDROME). MARVIN H. STRICK, J. Pediat. **24**:675 (June) 1944.

Four cases of fulminating meningococcemia are reported. Three patients were found at autopsy to have bilateral adrenal hemorrhage. The fourth patient recovered through the use of epinephrine, adrenal cortex extract, desoxycorticosterone acetate, saline solution and sulfathiazole.

Other cases from the literature are mentioned in which the patients recovered, and the treatments are listed.

Attention is called to similar cases in the literature in which autopsy failed to reveal the adrenal hemorrhages.

AUTHOR'S SUMMARY.

MEDICAL SOCIAL WORK IN AN EPIDEMIC OF POLIOMYELITIS. ALICE A. GRANT, J. Pediat. **24**:691 (June) 1944.

During the summer of 1943 southern Connecticut experienced a sizable epidemic of poliomyelitis, which, because of the number of victims, the violence of family reactions and community fear, had much in common with problems accompanying disasters. Most of the patients were cared for at New Haven Hospital. In trying to help families of patients to meet this emergency the medical social worker found there were certain recurring reactions of families toward the illness and its results. This article covers the medical social aspects of poliomyelitis, especially the reactions of the child patients, the adult patients and their families, as seen in case studies, to some of the specific problems created by epidemic conditions.

This epidemic in wartime that sent 172 patients to a civilian hospital produced an unusually severe emotional reaction both in the patients and in their families. Feeling about inability of the parents to see the physician, separation from the patient and fear of crippling ran like a refrain through the situations faced by each child's family. Because of epidemic conditions and war-induced shortages, it was impossible for the 152 families of sick children to see the patients or consult the physician in the usual manner. Though troubled, over four fifths of the number were able after interpretation to accept the recommended plan of treatment in spite of these limitations, thus indicating normal adaptability. One fifth of the group, however, had lower emotional thresholds and were able to carry out plans only with marked anxiety. Isolation from their child and inability to discuss the child's progress with the physician proved difficult for the parents. Seventy-one per cent seemed to accept these restrictions in view of the goals of treatment, while 29 per cent were less easily able to follow recommendations because their anxieties and fears were greater. Fear of crippling was implicit in every situation. Four fifths of the group could not articulate this fear, but one fifth verbalized it.

In contrast, case work with 13 adults with poliomyelitis disclosed less pressure of the feelings, a fact which may be accounted for largely by the availability of the physician to both patient and family and by the adult's ability to understand the implications of his illness himself, whereas the parents of the children had to take responsibility for thinking through the problem and making a decision for a dependent child.

It was obvious throughout the study that the more tense, anxious, unstable, immature persons found it more difficult to adapt themselves to the limitations and conditions necessitated by wartime epidemic shortages. In 96 per cent of the cases the recommended plan of treatment was accepted after resistances had been worked through; this indicates the validity of the teamwork, the methods and the technics used with these patients and their families.

An important element in the treatment of troubled families was the relief from financial worry made possible through financial help from the National Foundation for Infantile Paralysis.

The medical social worker's knowledge of and ability to organize and use community resources was a part of the service given.

The following recommendations are made with reference to future epidemics and disasters:

1. Preservation of the physician-patient relation is of first importance to reduce to a minimum dist and anxiety for the patient and the parents.

2. This experience shows that whenever isolatio a necessary precaution in hospitals arrangements sh be made for a limited type of visiting in which family can see, even if they cannot communicate w the patient.

3. The practice of interviewing every patient's fa should be continued and should be carried out b person with the skills of the medical social worker.

4. In order to meet the goal of individualized ser to each patient and to his family, all hospital serv must be flexible and adaptable and must work toget

GRANT, New Haven, Con

THE TREATMENT OF PERTUSSIS WITH LYOPHILE HY IMMUNE HUMAN PERTUSSIS SERUM. IRVING SCHEINBLUM, JESSE G. M. BULLOWA and GER WEISS, J. Pediat. **25**:49 (July) 1944.

Lyophile hyperimmune human pertussis serum w used in the treatment of 23 patients critically ill w pertussis. The severity of the illness in each case w judged by the patient's age, the severity of attacks paroxysms and the presence of cyanosis, apnea, em and pneumonic complications. The serum was obtai from the Philadelphia Serum Exchange. The tre ment consisted of three intramuscular injections 20 cc. of lyophile hyperimmune pertussis serum on th alternate days.

Sixteen of the 23 patients were under 1 year of a Twelve of these infants were in the 1 to 3 month group, and 75 per cent of them gave a good respon All the children under 12 months of age who w treated recovered. The 2 children who died were mo bund on admission to the hospital and did not rec a full course of treatment.

In 11 of the 23 patients the pertussis was complica by pneumonia, which was treated with sulfadiazine addition to the serum. There was no extension or r currence of the pulmonary involvement and no devel ment of pneumonia after a full course of treatment w the serum.

Not only was treatment with the lyophile hyperimm human pertussis serum effective early in the disease, b 2 patients treated in the eighth and ninth week of i ness, respectively, also gave good responses.

Agglutinin titers in the blood of 11 of the 23 patie were determined before treatment with the serum. I no instance were there any demonstrable agglutini in the blood. After the administration of the ser there were circulating agglutinins in the 11 patie

The lymphocytic blood counts of 15 of the 23 patie were studied before and after treatment with the ser In 12 of the 15 patients there was a substantial red tion in the number of lymphocytes after serum thera

AUTHORS' SUMMARY.

POLIOMYELITIS VIRUS IN THE HUMAN OROPHARYN HOWARD A. HOWE, HERBERT A. WENNER, DA BODIAN and KENNETH F. MAXCY, Proc. Soc. Exp Biol. & Med. **56**:171 (June) 1944.

Cotton swabs were rubbed on the oropharynx a the peritonsillar region of 14 patients during the fi week of poliomyelitis infection. The swabs were drop into a test tube with 1 cc. of sterile water and sto on solid carbon dioxide. Elution was accomplished b placing the swabs in phosphate buffer at p_H 8, and t

d was squeezed out of this cotton by placing it in yringe. After adjusting the preparation to p_H 6 and ting it with ether, which was later removed, injecns were made intracerebrally into 14 Rhesus monkeys ghing 8 to 10 pounds (3.6 to 4.5 Kg.) each. Typical omyelitis was produced in 7 of the animals, and alysis resulted in 6. Microscopic studies showed the racteristic lesions. The positive results were equally tributed among the paralytic and the nonparalytic jects.

HANSEN, Galveston, Texas.

RTHER OBSERVATIONS ON CONGENITAL DEFECTS IN INFANTS FOLLOWING INFECTIOUS DISEASES DURING PREGNANCY, WITH SPECIAL REFERENCE TO RUBELLA. C. SWAN, A. L. TOSTEVIN, H. MAYO and G. H. B. BLACK, M. J. Australia **1**:409 (May 6) 1944.

The authors found that of 12 mothers who contracted bella during pregnancy 10 bore children with conital defects. The abnormalities included cataract, f-mutism, heart disease, microcephaly and obliteration the bile ducts. With 3 exceptions (2 of them doubt-) all of the 10 mothers with congenitally defective ldren had contracted rubella within the first three nths of pregnancy.

GONCE, JR., Madison, Wis.

Acute Infectious Diseases

EUMATIC FEVER: DIET AS A PREDISPOSING FACTOR. DON CARLOS PEETE, Ann. Int. Med. **21**:44 (July) 1944.

Rheumatic fever and rheumatic fever disease result m a faulty diet and from lack of sunshine. The hor states that the typical diet of the rheumatic ient lacks food rich in vitamins A and D and espelly calcium, phosphorus and iron. The report is ed on a study of 50 patients with acute rheumatic er.

READING, Galveston, Texas.

LFATHIAZOLE AND STAPHYLOCOCCUS ANTITOXIN IN EXPERIMENTAL STAPHYLOCOCCIC INFECTION. EDWARD STEINFIELD, MICHAEL BRYLAWSKI and CARRIE B. NASH, J. Lab. & Clin. Med. **28**:1544 (Oct.) 1943.

Sulfathiazole administered perorally prolonged the e of mice infected with Staphylococcus aureus. The an time of survival of animals treated with sulfaiazole was thirteen days. The mean time of survival those treated with sulfathiazole and with staphylocus antitoxin was twelve and seven-tenths days, and those treated with serum alone six and five-tenths ys. The mean time of survival of controls was six d seven-tenths days. It is pointed out that experints with staphylococcic infections in animals do not tirely parallel clinical experience and that in the ter certain factors indicate that staphylococcic antixin may reenforce the immune processes of the host.

WILKINSON, Galveston, Texas.

RSISTENCE OF INFLUENZA VIRUS ON THE HUMAN HAND. ERNESTINE R. PARKER and WARD J. MACNEAL, J. Lab. & Clin. Med. **29**:121 (Feb.) 1944.

Experiments to determine the time of survival of inenza virus of the Melbourne strain on the human nd are described. These studies show that potent fluenza virus may remain active for forty-five minutes the dry state on the hand. A solution of soap tended inactivate the virus on the skin. The possibility of manual transmission of influenza, especially by handlers of food, is pointed out.

WILKINSON, Galveston, Texas.

DEVELOPMENT OF ANTIBODY FOLLOWING VACCINATION OF INFANTS AND CHILDREN AGAINST PNEUMOCOCCI. HORACE L. HODES, JAMES F. ZIEGLER JR. and HELEN D. ZEPP, J. Pediat. **24**:641 (June) 1944.

Among a group of children over 2 years of age the intradermal inoculation of a dose of heat-killed type I pneumococci which did not cause systemic reactions or serious local reactions was followed in nearly every instance by an abrupt rise in the serum mouse-protective antibody titer. The level of antibody attained was of the same order of magnitude as has been reported in adults after treatment for type I pneumococcic pneumonia with a sulfonamide compound. The rise in antibody titer which followed vaccination persisted for at least several months.

In contrast with this result, intradermal inoculation with type I pneumococcic vaccine did not produce a rise in antibody titer in infants under 2 years of age.

AUTHORS' SUMMARY.

THE THERAPEUTIC VALUE OF SULFAGUANIDINE IN THE TREATMENT OF BACILLARY DYSENTERY AT AN AUSTRALIAN GENERAL HOSPITAL. B. A. BAKER, M. J. Australia **1**:435 (May 13) 1944.

Twenty-six young adult patients with bacillary dysentery were treated with sodium sulfate (three doses of 60 grains [3.83 Gm.] each at intervals of an hour) and then with sulfaguanidine, and 31 patients were treated with sodium sulfate alone (60 grains [3.83 Gm.] every four hours for one day and then every eight hours for from four to six days). The initial dose of sulfaguanidine was 7 Gm., followed by 3.5 Gm. every four hours for one or two days and then every eight hours for six or seven days. Every patient treated with sulfaguanidine showed a rapid reduction in the number of stools passed during the early stage of treatment and rapid improvement in his clinical condition. Patients under sulfaguanidine therapy had less toxemia, quick increase of appetite and rapid loss of abdominal discomfort and tenderness. The average number of days before sigmoidoscopic findings became normal was seventeen and five-tenths for the sulfaguanidine-treated series and twenty-four and four-tenth days for the sodium sulfate-treated series. No toxic symptoms were observed, nor was any rash apparent which could be attributed to sulfaguanidine.

GONCE JR., Madison, Wis.

Chronic Infectious Diseases

PROGNOSIS IN WHITE AND COLORED TUBERCULOUS CHILDREN ACCORDING TO INITIAL CHEST X-RAY FINDINGS. MIRIAM E. BRAILEY, Am. J. Pub. Health **33**:343 (April) 1943.

This paper discusses the extent of pulmonary lesions demonstrated on initial roentgen examination of 1,148 children studied in the outpatient clinic for tuberculous children of Johns Hopkins Hospital, Baltimore, since 1928. Their subsequent course is also analyzed.

This clinic admits infants under 2 years of age who have positive reactions to tuberculin and any of their siblings who show a positive reaction. Of the total number studied, the result of the tuberculin test was not recorded for 14, and 7 others had negative reac-

tions. All of these 21 children either had healed pulmonary lesions or died of tuberculosis, usually shortly after coming under observation. The study comprised 404 white children and 744 Negro children. About 60 per cent were under 5 years of age. Thirty per cent were between 5 and 9, and 10 per cent were between 10 and 14 years of age. About one fifth of the children were brought to the dispensary because of some complaint; the remainder were checked because of known tuberculosis in the family. All children were members of families unable to pay for medical care, and the great majority were supported by public relief. All but a small number received only dispensary supervision during the most active phase of their tuberculous infection. The author estimates that the duration of exposure to the primary patient in the family was longer for the white children, but she cannot estimate the intensity of exposure.

The results of the initial roentgen examination in each case were classified in one of four groups: No evidence of tuberculosis; nodal lesions at the mediastinum, parenchymal lesions and reinfection lesions. In general, the younger the child the more liable he was to have a recognizable pulmonary lesion and the more likely it was to be a serious one. The decrease in frequency and severity of pulmonary lesions with increasing age was greater among white children. Under the age of 1 year only about 30 per cent of the children had roentgenograms showing no evidence of tuberculosis and almost 50 per cent had parenchymal lesions. For the age group 10 to 14, 71 per cent of the white children and 56 per cent of the Negro children had no roentgenographic evidence of the disease. For every age group after the first year of life a greater percentage of parenchymal lesions was found in the Negro children. More than 5 per cent of the Negro children in the age groups 5 to 9 and 10 to 14 had parenchymal lesions. Of the white group between 5 and 9 less than 1 per cent had parenchymal lesions, and none was found in the group aged 10 to 14. Nodal disease was found in 29 per cent of the white children and in 26 per cent of the Negro children. One white child and 5 Negro children had reinfection tuberculosis. It is of interest that during the course of the study reinfection tuberculosis developed in 19 other Negro children but in no other white children.

Among children who were under the age of 3 when first found to react positively to tuberculin, there was a mortality of 8.1 per cent for the white children and 19.8 per cent for the Negro children in the next ten years. More than half the deaths occurred in the first year; all the deaths of white children and most of those of Negro children occurred within the first five years. Approximately 21 per cent of the white children and 36 per cent of the Negro children with parenchymal lesions at the time of the first roentgenogram were dead at the end of ten years. Approximately 4 per cent of the white children and 8 per cent of the Negro children with no parenchymal lesions at the time of the first roentgenogram were dead at the end of ten years.

Two hundred and twenty-six white and 410 Negro children were between 3 and 15 years of age when first discovered to be infected. Among the white children there was only 1 death (from the group of 4 with parenchymal lesions), which occurred within the first year of observation. Among the Negro children there were 12 deaths (3.0 per cent) within one year and 23 deaths (5.6 per cent) within ten years. Of the 410 Negro children 37 had parenchymal disease and 5 had reinfection lesions at the time of the initial roentgenogram. Thirteen of the infants with parenchymal disease and 4 of those with reinfection lesions were dead at the end of ten years. The other 6 who died were from the group of 368 children who had no lesions or only nodal lesions at the time of the initial roentgenogram.

For the entire group there were 73 deaths among children discovered to be infected before the age of 15. Approximately 40 per cent of these deaths were due to miliary tuberculosis, and 40 per cent were due to tuberculous meningitis. There were 24 deaths of children who were between 3 and 15 years of age at the time of discovery. Approximately 15 per cent of these deaths were due to miliary tuberculosis, 25 per cent to tuberculous meningitis and 40 per cent to reinfection tuberculosis.

PLATT, Rochester, N. Y.

CONGENITAL TUBERCULOSIS: TUBERCULOSIS STUDIES IN OFFSPRING OF MOTHER GUINEA PIGS HEAVILY INFECTED INTRAVENOUSLY. H. J. CORPER and MAURICE L. COHN, Am. Rev. Tuberc. **48**:25 (July) 1943.

The intravenous injection of a relatively large dose of highly virulent human tubercle bacilli into pregnant guinea pigs did not result in the passage of bacilli to the fetuses for at least nine to as long as twelve days in spite of the fact that the mothers succumbed within one or two weeks after the birth of the offspring.

No discernible specific tuberculoimmunity or specific tuberculoallergic hypersensitiveness developed in the offspring of such heavily infected mothers.

In guinea pigs given intravenous injections of viable avirulent human tubercle bacilli, definite specific tuberculoallergic hypersensitiveness to tuberculin and excellent specific tuberculoimmunity developed. No evidence was noted that this hypersensitiveness to tuberculin or this tuberculoimmunity was transmitted to the offspring.

SMITH, Ogden, Utah.

THE CHEMISTRY OF THE LIPIDS OF TUBERCLE BACILLI. R. J. ANDERSON, R. E. REEVES, M. M. CREIGHTON and W. C. LOTHROP, Am. Rev. Tuberc. **48**: (Aug.) 1943.

Tuberculous human lung tissue was examined for certain chemical compounds, namely, phthiocol, tuberculostearic acid, phthiotic acid and the specific polysaccharides which are present in the human tubercle bacillus when it is grown on a synthetic medium. It was impossible to detect even traces of these substances in the tuberculous lung tissues examined, although the first three substances when incorporated with normal human lung tissue could be isolated and identified, a fact which shows that these compounds are not destroyed by lung tissue.

SMITH, Ogden, Utah.

AN EARLY PRIMARY TUBERCULOUS PULMONARY FOCUS ABOUT FOUR TO EIGHT WEEKS OLD. WALTER PAGEL and DOROTHY STOPFORD PRICE, Am. Rev. Tuberc. **48**:614 (June) 1943.

This, the fourth recorded early primary tuberculous focus, was found in a girl who died at the age of days. The mother and the elder sister of the infant had advanced pulmonary tuberculosis, and she was watched carefully for the development of primary tuberculosis. The first evidence of infection, a positive reaction to the Mantoux test with tuberculin in

tion of 1:100, was noted three days before her th. In a focus of 0.9 by 1.2 mm. in the lower lobe the right lung the following changes were found: patchy alveolitis of the desquamative macrophago- ic type and (*b*) caseation, which was restricted to alveolar plugs in the center of the focus. A strong ltration of lymphocytic and mononuclear cells into septums, with acid-fast rods, was observed.

SMITH, Ogden, Utah.

Chronic Contagious Diseases

E LABORATORY DIAGNOSIS OF TUBERCULOSIS. MELVIN E. KOONS, J. Lab. & Clin. Med. **29**:75 (Jan.) 1944.

This is a report of a comparative study of animal culation and of microscopic examination for tubercle illi. In the 590 specimens examined animal inocula- n proved far superior to microscopic examination.

WILKINSON, Galveston, Tex.

MPARATIVE RESULTS WITH TRANSDERMAL (OR TRANSCUTANEOUS) AND INTRACUTANEOUS TUBERCULIN TESTS. H. J. CORPER, J. Lab. & Clin. Med. **29**: 398 (April). 1944.

The author reports the results of a transdermal erculin test (prepared from an autolytic tuberculin) compared with the results of an intradermal test th a purified protein tuberculin (prepared from the me original bacillary cultures). Among 103 patients ted 90 (87 per cent) gave positive reactions to the nsdermal test and 89 (86 per cent) gave positive actions to the intradermal test. Agreement between e two tests occurred with 82 of the positive tests. The author emphasizes the importance of the tuberlin test as one of the best biologic diagnostic tests.

WILKINSON, Galveston, Tex.

OMIN IN THE TREATMENT OF TUBERCULOUS MENINGITIS. WILLIAM J. MORROW, HAROLD C. EPSTEIN and JOHN A. TOOMEY, J. Pediat. **24**:623 (June) 1944.

Eleven patients with tuberculous meningitis were ated with promin, the sodium salt of p,p′-diaminophenylsulfone-N,N′, didextrose sulfonate, without any neficial result in either prolonging or maintaining e or in altering the course of the disease.

TOOMEY, CLEVELAND.

HE CARE OF THE SYPHILITIC CHILD. JOSE ALBERTO PRADERI and MARIO TAGLIORETTI, Arch. de pediat. d. Uruguay **14**:611 (Oct.) 1943.

This article deals with the need for close cooperaon of all agencies concerned with the public health pects and the medical aspects of congenital syphilis d stresses close cooperation between pediatricians, philologists and social workers in the recognition and anagement of this condition.

PLATOU, New Orleans.

ONTRIBUTION TO THE STUDY OF THE INDEX OF INFECTION AND OCCURRENCE OF TUBERCULOSIS IN INFANCY. G. H. MIYARES and R. O. PEDRAZA, Bol. Soc. cubana de pediat. **15**:1 (Jan.) 1943.

This study is based on 5,000 children attending the almette Dispensary in the city of Habana, Cuba. Of the total number, 2,779, or 55.58 per cent, reacted positively to the Mantoux test. The number of reactors among those without evidence of active tuberculosis, however, was 45 per cent. The proportion of positive reactions was greater among the Negroes and the half-breeds than among the white children, being 62.14 per cent, 56.84 per cent and 53.93 per cent respectively.

KEITH, Rochester, Minn.

Diseases of Blood, Heart and Blood Vessels and Spleen

STANDARDIZATION OF SEDIMENTATION RATE. J. W. CUTLER, Am. Rev. Tuberc. **48**:314 (Nov.) 1943.

The variety of technics and methods for expressing the results of sedimentation tests continues to cause confusion. Cutler attempts to clarify the problems involved by proposing a technic based on the three phases of the phenomena of sedimentation and the mechanical factors involved in the process.

The role of the erythrocytes in sedimentation is a passive one. The composition of the plasma alone is responsible for the arrangement of the red cells into rouleaux. Complete parallelism exists between the size of the rouleaux and the sinking velocity of the cells; The larger the rouleaux the more rapid the settling, and vice versa. Increased settling of the erythrocytes is brought about by changes in the composition of the plasma, the result of absorption into the blood stream of certain as yet unknown products of the breakdown of tissues. This alteration in the composition of the plasma is responsible for the size of the rouleaux; hence the rapidity of sedimentation.

The process of sedimentation occurs in three phases:

1. Aggregation, or formation of rouleaux. This period varies from five to fifteen minutes, occasionally lasting longer.

2. Settling. Once formed, the rouleaux settle at uniform speed, more or less in accordance with Stokes's law of hydrodynamics: The larger the aggregate, the greater the mass and the faster the settling, and vice versa.

3. Packing. This occurs as the rouleaux approach the bottom of the tube and their rate of settling becomes progressively slower. In this phase the phenomena of sedimentation should be considered to have ceased.

The second phase is the only one of clinical significance, i. e., the period when the rouleaux are fully formed and are settling at a constant velocity.

The technic advocated is the use of the Cutler tube of a capacity of 1 cc. graduated into 50 mm. divisions. One-tenth cubic centimeter of 3.8 per cent sodium citrate and 0.9 cc. of blood are introduced into the tube. The sedimentation column is read every five minutes for one-half hour. The maximum settling in any five minute period is the sedimentation rate. A maximum settling of 1 mm. or less is normal. Generally rates up to 3 mm. represent disease with slight activity, rates up to 8 mm. disease with moderate activity, and higher rates disease with marked activity.

EUGENE H. SMITH, Ogden, Utah.

BIOPSY OF BONE MARROW PERFORMED BY A NEW AND SIMPLE INSTRUMENT. HENRY TURKEL and FRANK H. BETHELL, J. Lab. & Clin. Med. **28**:1246 (July) 1943.

The advantages and limitations of securing bone marrow for biopsy by aspiration are discussed. The limitations have led many hematologists to rely on examination of material obtained by trephine. The authors describe an instrument designed to obtain a small specimen of marrow with no more effort or discomfort than is involved in aspiration. The instrument consists of two needles with stylets, an outer guiding needle of 14 gage and an inner trephine needle of 17 gage. Photomicrographs of section of marrow secured by this method are presented.

WILKINSON, Galveston, Texas.

THE PROPHYLACTIC USE OF SULFANILAMIDE IN CHILDREN WITH RHEUMATIC HEART DISEASE. CHARLES R. MESSELOFF and MILTON H. ROBBINS, J. Lab. & Clin. Med. **28**:1323 (Aug.) 1943.

Sulfanilamide in daily doses of 1.2 Gm. was administered to 25 ambulatory rheumatic children. Thirty children with rheumatic heart disease who were not given sulfanilamide were used as controls. Observations were carried out during 2 periods of eight months each. Minor toxic effects from sulfanilamide were noted: anorexia, moderate decrease in the level of hemoglobin in the blood, moderate leukopenia and mild maculopapular eruptions. Two deaths and 1 rheumatic recrudescence occurred in the treated group and 1 death and 2 recrudescences in the control group. Although the series of cases was small, indications were that sulfanilamide is not of value in preventing rheumatic recrudescences.

WILKINSON, Galveston, Texas.

Diseases of Nose, Throat and Ear

A BACTERIOLOGIC, CLINICAL AND THERAPEUTIC STUDY OF ACUTE SUPPURATIVE OTITIS MEDIA IN INFANCY. A. AUGUSTO LATORRE and P. FRANCISCO LANDA, Rev. de otorrinolaring. **3**:63 (Sept.) 1943.

Latorre and Landa made a clinical study of acute suppurative otitis media in infancy. The bacteriologic analysis revealed the incidence of pathogenic microorganisms to be: Streptococcus haemolyticus 59.3 per cent, Staphylococcus 25 per cent, Pneumococcus 22.3 per cent and Pneumococcus type III 7.9 per cent.

In treatment they were extremely conservative, depending almost entirely on early paracentesis, irrigation and application of external heat. In this group no chemotherapy was employed. As to results, 78.3 per cent of the ears were cured without any complications; in 12.5 per cent the otitis became chronic; in 2.8 per cent symptoms referable to the mastoid process developed. Mastoidectomy was required in only 2 cases.

The authors stress the importance of early paracentesis, particularly in cases in which there is doubt as to the presence of a latent process. They also believe that small blood transfusions are of extreme value in combating the infection. They depreciate the value of the use of sulfonamide compounds.

They studied a group of 45 cases in which sulfonamide compounds were employed, and they had a control group of 50 cases in which the patients did not receive any chemotherapy. The results in each group were almost identical as to the duration of the discharge, the number of cases in which the otitis eventually became chronic and the number that ultimately required mastoidectomy.

PERSKY, Philadelphia. [ARCH. OTOLARYNG.

Diseases of Lungs, Pleura and Mediastinum

PURULENT PERICARDITIS COMPLICATING PNEUMO... MOSES COOPERSTOCK, J. Pediat. **24**:656 (June) 1...

An instance is reported of recovery from purul... pericarditis by an 8 month old infant after a si... large therapeutic aspiration (135 cc. of pus) of ... pericardial sac and the subsequent development of pn... mopericardium, factors which appear to have opera... together in bringing about the favorable outcome. ... development of pneumopericardium may have been ... either spontaneous or accidental origin, although ac... dentally induced pneumopericardium as a result of ... ploratory or therapeutic pericardial paracentesis is ... relatively uncommon occurrence. The presence of pn... mopericardium may have been primarily responsible ... the failure of the exudate to reaccumulate.

Pericardial paracentesis alone as a therapeutic m... sure for purulent pericarditis has proved uniformly ... successful, surgical drainage by means of pericardiot... providing the far superior method of treatment (50 ... cent of cures has been reported). The suggestion ... derived from the events in this case that therape... aspiration plus induced pneumopericardium may se... as a conservative method of treatment in purulent pe... carditis when expert surgical skill is not readily av... able. The inclusion of penicillin in the treatment m... be of additional value

COOPERSTOCK, Marquette, Mich.

PNEUMONIA, PNEUMOTHORAX AND EMPHYSEMA F... LOWING INGESTION OF KEROSENE. E. P. SCOTT... Pediat. **25**:31 (July) 1944.

The author reports an instance of ingestion of ke... sene complicated by bilateral bronchopneumonia, pn... mothorax and emphysema of the soft tissues of t... chest.

Aside from the administration of sulfathiazole, ... treatment was symptomatic. Recovery was uneventf...

SCOTT, Louisville, Ky.

A REVISED TECHNIC FOR THE TREATMENT OF EMPYE... EDWARD J. MCGRATH, South. M. J. **37**:127 (Mar... 1944.

A revision of the technic for the treatment of clos... drainage of the thoracic cavity is described in det... The tube is said to be comfortable to the patient a... to allow adequate drainage. The technic has be... used on both children and adults at the University H... pitals in Cincinnati for four years.

LOGAN, Rochester, Minn.

SPONTANEOUS PNEUMOTHORAX, MASSIVE COLLAPSE A... SUBCUTANEOUS EMPHYSEMA COMPLICATI... ASTHMA. C. ELAINE FIELD, Arch. Dis. Childh... **18**:197 (Dec.) 1943.

A case is reported of a girl aged 4 years and 2 mon... who showed the combined features of asthma, subc... taneous emphysema, collapse of the left lung and spo... taneous pneumothorax in the right side of the ch... with recovery. This is probably a unique phenomen...

KELLY, Milwaukee.

ORTANCE OF THE LATERAL ROENTGENOGRAM IN PNEUMONIA OF THE MIDDLE LOBE OF THE LUNG. RAFAEL DE LA PORTILLA Y LEVASTIDA, Bol. Soc. ubana de pediat. **15**:38 (Jan.) 1943.

With the aid of the lateral roentgenogram the author demonstrated the greater frequency of pneumonia the middle lobe of the lung. He states that the e of the shadow of such pneumonia is always gular, that its origin is always toward the periphery that the route of infection is most likely hematog-is.

KEITH, Rochester, Minn.

Nervous Diseases

THE MODE OF REPRESENTATION OF MOVEMENTS IN THE MOTOR CORTEX, WITH SPECIAL REFERENCE TO "CONVULSIONS BEGINNING UNILATERALLY" (JACKSON). F. M. R. WALSHE, Brain **66**:104, 1943.

Walshe points out that the first indication of a resentation of movement and of function in the bral cortex was deduced from clinical observation. clinical neurologist today, viewing the knowledge hand regarding the nature of the cortical represen-on of movements, finds two apparently unrelated ces of information: (1) the literature on electrical ulation of the cortex, presenting the picture of a aic of excitable points, each yielding a character-motor response; this excitable cortex being un-le, as is evidenced by facilitation, deviation and rsal; and (2) the clinical phenomena of jacksonian with their three predominant foci in the motor ex, resulting in movements of the thumb and the x finger, the angle of the mouth and the hallux. he literature on experimental physiology of the or cortex has been primarily oriented along ana-ic lines; that is, it has been more concerned with "where" than with the "how" of the cortical repre-tation of movement. Jackson pointed out that the bral cortex represents sensorimotor processes and the performing parts. That muscles themselves not represented in the cortex is indicated by the mon clinical experience of paralysis of the exten-s of the wrist in voluntary extension, with the sence of powerful synergistic contraction in forced sping. Through study of the movements elicited electrical excitation or by disease processes of the tex, the nervous mechanisms involved in a given vement have been determined, and so the movement come to be considered localized, or represented. ilding up on this basis produced general principles cortical representation and of the mode in which cortex initiates movement. Thus used, "represen-ion" means all those processes in the cortex by ich these visible results are brought about.

However, according to Jackson's hypothesis, the tor cortex represents all the movements which an ividual organism is capable of initiating. Thus, a ttern of excitation must exist for each purposive vement, and the leading motile parts must have the st extensive representation; the greater the spatial tension of representing cortical structures, the more mplete must be the integration. The motor cortex st be considered not as a mosaic of abrupt localiza-ns but as a complex pattern of overlapping and aded representations. Therefore, after a focal lesion, ile the functions of the destroyed area are not taken er by other areas, the vast repertory of movement t may allow the functional deficit to be hidden; on the contrary, a stimulating lesion may release a widespread convulsion without necessarily a wide distribution of the excitatory process through the cortex.

Walshe points out the difficulty in reconciling the theories of punctate localization with the phenomena of facilitation, deviation and reversal of response, but on the basis of Jackson's hypothesis of cortical representation these phenomena are accounted for by the changes in the relative thresholds of excitability in the different patterns of excitation. Each focus in the motor cortex has through neuronal connections the substratum of many patterns of excitation, but one of these is primary. Appropriate antecedent local stimulation will bring about facilitation by lowering the threshold of excitability, so that the change is not spatial but is due to deviation of response. Therefore deviation, as well as augmentation and reversal, of response, is a consequence of facilitation. Coordination in time, resulting in the orderly sequence of movement, probably has as its primary factor facilitation. A destroying lesion of the cortex is followed both in man and in experimental animals by partial return of function. This, Walshe contends, is inexplicable on the basis of the theory of punctate localization but is consistent with Jackson's hypothesis: Moving parts must have wide representation in the cortex, and cortical points must contain many representations.

Walshe reviews the clinical features involved in convulsions beginning unilaterally and indicates those which tend to corroborate Jackson's hypothesis of representation and the foregoing correlation of that hypothesis with present neurophysiologic observations. Since a convulsion may spread although cortical discharge may remain restricted and since the patterns of unilateral seizures beginning in two different peripheral parts differ, Walshe concludes that the field of convulsion in the musculature is probably always wider than the field of cortical excitation. The compound order of spread of a unilateral seizure (increase in intensity as well as in distribution) is explicable on Jackson's hypothesis but not on the theory of punctate localization. The onset of jacksonian seizures in one of three areas is not explicable on the basis of the puncture theory but can be accounted for only by Jackson's hypothesis, and these three "leading parts" probably have the widest fields of low threshold excitability.

The spread of unilateral seizures is not always consistent with the topographic charts of the motor cortex; these exceptions are consistent with the jacksonian concept of representation. Jackson maintained that movements of both halves of the body were represented in the motor cortex of each hemisphere, and so focal convulsions from one motor cortex could become generalized. Walshe cites as a parallel the bilateral neurologic signs frequently observed in cases of severe hemiplegia. Focal seizures characterized by sudden transient motor impairment or loss of speech may afford confirmation of the presence of inhibitory areas of the cortex. The same mechanism may explain the postseizure flaccid coma.

FORSTER, Philadelphia.
[ARCH. NEUROL. & PSYCHIAT.]

Psychology and Psychiatry

EFFECTS OF WAR ON THE MINDS OF CHILDREN. BERT I. BEVERLY, Am. J. Pub. Health **33**:793 (July) 1943.

The essentials for mental health are stated to be a feeling of security (as to parents, family, school, church, community and nation) and an opportunity to grow

according to individual patterns. The author points out that the aspects of the war effort which may impair a feeling of security or interfere with individual development depend on the age of the child. The infant is upset by an anxious parent; the adolescent by a feeling that he is not contributing adequately to the war effort or that there will be no opportunity for him in the postwar era. Specific means of minimizing the effects of the war and of helping children are discussed.

PLATT, Rochester, N. Y.

Diseases of the Genitourinary Tract

A DIAGNOSTIC AID IN PERINEPHRIC ABSCESS. GEORGE G. GILBERT and JOHN E. DEES, South. M. J. **37**:438 (Aug.) 1944.

The authors suggest that the diagnosis of perinephric abscess may be made earlier in many cases of apparently uncomplicated renal infection by considering inadequate response to therapy with sulfonamide compounds as a diagnostic aid. Three cases are presented.

LOGAN, Rochester, Minn.

MARCH HEMOGLOBINURIA: DESCRIPTION OF THE FEATURES OF THIS CONDITION AND REPORT OF A CASE. L. M. BRYCE, M. J. Australia **2**:49 (July 15) 1944.

The salient features of march hemoglobinuria are the following: 1. It occurs in boys and young men, usually in the second or third decade. 2. It is related to exercise in the upright position but is not necessarily associated with lordosis. 3. Constitutional symptoms are absent or mild. 4. The course is benign. The condition appears to have no permanent ill effects and progresses without specific treatment toward ultimate spontaneous recovery, which usually takes place within a year or two after the initial attack. March hemoglobinuria must be differentiated from other forms of hemoglobinuria of paroxysmal occurrence such as (1) blackwater fever, (2) hemoglobinuria due to the ingestion of certain drugs and foodstuffs, (3) cold hemoglobinuria, which almost always has a syphilitic basis, (4) nocturnal hemoglobinuria, which is associated with persistent and progressive hemolytic anemia and which ultimately has a fatal outcome and (5) paralytic myohemoglobinuria. A typical case is described.

It is pointed out that the usual investigation of hematuria or hemoglobinuria is likely to be carried out with the patient at rest. Hence hospitalization for investigation will actually tend to prevent the occurrence of march hemoglobinuria and so fail to reveal the symptoms of which the patient complains. Unless, therefore, the possibility of this condition, which in its severe forms is rare, is kept in mind, considerable difficulty and delay may occur in confirming the patient's statements and establishing the diagnosis.

GONCE, Madison, Wis.

Diseases of the Ductless Glands; Endocrinology

THE EXCRETION OF GONADOTROPHIC HORMONE BY PREPUBERAL AND ADOLESCENT GIRLS. H. R. CATCHPOLE and W. W. GREULICH, J. Clin. Investigation **22**:799 (Nov.) 1943.

Data on the daily urinary excretion of pituitary gonadotropic hormone by 12 girls between the ages of 4½ and 14 years indicate that the picture of pituitary excretion of gonadotropic hormone during the periods of prepuberty and adolescence represents a stage tween the quiescence characteristic of early childh and the fully developed adult cycle. Absent from urine of the youngest girls, the hormone appeared du preadolescence in increasing amounts but with fluc tions in the daily amounts; the fluctuations did appear to be systemic or cyclic. The pattern of ou of gonadotropic hormone appeared to be related m closely to the developmental status than to the chro logic age of the subjects.

GUEST, Cincinnati

THE UTILIZATION OF INTRAVENOUSLY INJECTED S IN NORMALS AND IN PATIENTS WITH CUSHI SYNDROME BEFORE AND AFTER ADMINISTRATION DESOXYCORTICOSTERONE ACETATE. LOUIS J. SOF GERSON LESNICK, S. ZELIG SORKIN and H SOBOTKA, J. Clin. Investigation **23**:51 (Jan.) 1

The utilization of intravenously injected salt b before and after administration of desoxycorticoster acetate was studied in 12 normal persons and i patients with Cushing's syndrome. In normal pers the injection of the hormone caused a decrease in urinary excretion of sodium and of chloride ions compared with the control values. In the patients w Cushing's syndrome the injection of the hormone sulted in an increase in the urinary excretion of sodi and of chloride ions. The authors suggest that the may be of value in the diagnosis of hyperfunction the adrenal cortex.

GUEST, Cincinnati

JUVENILE DIABETES INSULIN SENSITIVITY. HENRY FEINBLATT, BARNETT B. ALPERT and EDGAR FERGUSON JR., J. Lab. & Clin. Med. **29**:366 (Apr 1944.

Reports of cases are presented describing exacer tions of the diabetic state in young patients. Th exacerbations occurred independently of the dietary in spite of daily medication with insulin and are co sidered to be due to increased activity of the anter lobe of the pituitary gland. The normoglycemic t of case is also presented; this type shows perio glycosuria, followed by definite and permanent diabe mellitus. The authors suggest that the use of insu during the normoglycemic phase might be helpful preventing a permanent diabetic state.

Also, reports of cases of juvenile diabetic patie observed before and throughout their pregnancies a presented. In each case there was dramatic sensitiv to insulin during the immediate postpartum peri In 1 case the accustomed dose of insulin resulted fatal hypoglycemia. The authors suggest that the mech nism here is the diabetogenic factor of the anter lobe of the pituitary gland which may come from t posterior lobe of the pituitary gland.

WILKINSON, Galveston, Tex.

THE FREE DIET IN JUVENILE DIABETES. JAMES BRUCE, South M. J. **37**:34 (Jan.) 1944.

The "free' diet is recommended for juvenile diabe patients because it requires less regimentation than t stricter diets and makes them feel less different fr their fellows. It can be used early, after the childre and their parents have had a period of training w weighed or measured diets and understand the dange of diabetes. It may be instituted then with the ho that the children and their parents can thereafter ma age the disease by "ear." It should be used only patients having intelligence, judgment and self contr

e author prefers the use of protamine insulin to of regular insulin because only one daily injection e former is required. It controls glycosuria suffi- y well for practical purposes; he feels that glyco- is of little harm as long as no evidence of acidosis s.

LOGAN, Rochester, Minn.

Diseases of the Muscles, Bones, Joints and Lymph Glands

OGENESIS IMPERFECTA AND OSTEOPSATHYROSIS. S. OSENBAUM, J. Pediat. **25**:161 (Aug.) 1944.

Osteogenesis imperfecta of the Vrolik type and psathyrosis of the Lobstein type appear to have a mon pathogenesis and to represent different mani- tions of a single disease.

Osteopsathyrosis may develop after cerebral dis- rs and presumably in response to them.

Osteosclerotic proliferation of the margins of the bral bodies has been observed in a case of osteo- yrosis.

Treatment of osteopsathyrosis with extract of the ior lobe of the hypophysis is recommended.

AUTHOR'S SUMMARY.

NT EXPERIMENTAL STUDIES ON THE EFFECTS OF MMOBILIZATION OF DENERVATED MUSCLES. Qra EONARD HUDDLESTON, South. M. J. **37**:72 (Feb.) 944.

onflicting ideas exist regarding the proper treatment care of denervated muscles. Particularly is this with respect to patients having anterior poliomye-

experiments performed on dogs the author obtained following results:

Lack of immobilization of the denervated muscles not interfere with regeneration of the motor nerve ply.

Regeneration of the motor nerve supply was more plete in the animals in which no immobilization was lied after denervation than in those in which the rvated muscles were immobilized.

Immobilization in some way interfered with the nervation of denervated muscles.

Muscles immobilized in the position of maximal gth of muscle showed less functional recovery than e immobilized in the position of minimal length.

LOGAN, Rochester, Minn.

UNUSUAL OSSEOUS DYSTROPHY. JAMES F. BRAILS- FORD, Arch. Dis. Childhood **18**:98 (June) 1943.

An example of osseous dystrophy is described, char- erized by dense epiphyses which fused early, though elopment of other ossific centers was markedly de- ed. Grotesque dwarfing, with apparently normal in- igence, was associated with severe spinal curvature bilateral coxa vara.

KELLY, Milwaukee.

Diseases of the Eye

LOBOMA OF THE OPTIC NERVE: REPORT OF A CASE. T. STEINBERG, Am. J. Ophth. **26**:846 (Aug.) 1943.

A man aged 20 had visual acuity of 6/60 in the left , which was unimproved by a correction of —1 D. . ⊂ — 1 D. cyl., axis 55. There appeared to be a pseudo optic disk, about three times the size of the normal disk. Its center was conspicuously excavated, from —6 to —8 D.

The central portion was bluish gray and the periphery grayish white. The margins were sharply defined, the temporal side showing a thin, irregular ring of choroid pigment. The vessels appeared to emerge at the borders and bend sharply at the edges, corresponding to group 3 of Caspar's classification. No other lesions were present. The nasal field showed slight peripheral contraction and decided enlargement of the blindspot.

ZENTMAYER. [ARCH. OPHTH.]

HEREDITARY CORNEAL DYSTROPHY. J. R. MUTCH, Brit. J. Ophth. **28**:49 (Feb.) 1944.

The subject of hereditary corneal dystrophy is considered fairly exhaustively. It is pointed out that Groenouw first described the disease in 1890 under the term "nodular corneae." That the disease is familial was first noted by Fuchs, in 1901. Mutch advocates the use of the descriptive term "granular" dystrophy and suggests that the term "nodular" be abandoned because it has caused so much confusion. He gives a pedigree in which 24 persons were affected. Of the affected, 14 were males and 10 females; the youngest was a boy aged 12 and the oldest was 90 at the time of examination. Eight of the affected persons were over 60 when examined. Fourteen of the patients were myopes. Mutch is inclined to think that the low myopia or myopic astigmatism was due in some cases to the corneal lesions but that in others the myopia was due also to heredity and was connected with the corneal disease. Up to the age of 50 corrected vision was in no instance less than 6/12. The visual acuity, however, does not suffer an irresistible diminution with the advance of age, as 4 affected patients, well over 60, still have vision of 6/12 or better. Only 1 patient, a man over 90, could have been certified as blind. In an old granite polisher the corneas had the appearance of hereditary dystrophy. The lesions, however, were all on the surface, but the appearance of the eye and the distribution of the opacities were exceedingly similar. The lesion is not granular corneal dystrophy if it is unilateral, if it shows signs of vascularity, if opacities are visible on examination with the naked eye in a patient under 30 and if there is absence of a similar condition in both parents. The author considers the condition as probably a nutritional disturbance. The cornea receives its nutrition from the vascular loops at the limbus, lymph permeating centrally between the layers of the corneal lamellas. Any error in this circulation of lymph would affect the area farthest from the supply, namely, the central region of the cornea. Weight is given to this argument by the observation of Bucklers and other investigators who stated that considerable improvement followed an intercurrent inflammation with vascularization of the cornea.

The progress of the disease was not brought to a standstill with certainty under any form of treatment used; far less was the disease made to retrogress. In certain cases the attempt at cure was more harmful than the disease.

The refractive error should be corrected by glasses and the refraction periodically checked. The general health should be kept at as high a level as possible, with careful attention to diet, which should be supplemented by the periodic addition of vitamins A and B.

The article is illustrated, and an excellent bibliography is appended.

ZENTMAYER. [ARCH. OPHTH.]

Skin Diseases; Allergy

EYE MANIFESTATIONS OF XERODERMA PIGMENTOSUM. A. B. REESE and I. E. WILBER, Am. J. Ophth. **26**: 901 (Sept.) 1943.

The authors state that xeroderma pigmentosum, although primarily a disease of the skin and mucous membranes, is frequently marked by ocular complications. They give the following summary of their article:

The cases of 4 patients having xeroderma pigmentosum with ocular complications are reported, including the results of microscopic examination of one eye in which an epithelioma of the cornea developed at the site of an ulcer resulting from exposure. One patient died at the age of 7 years and 1 at the age of 11 years, but the other 2 patients have survived up to the time of writing, to the ages of 16 and 30 years. Three of the patients were siblings. From the ophthalmologic standpoint, the disease affects primarily the lower lid, producing an atrophy which begins at the margin and gradually progresses, so that ultimately the entire lower lid may disappear. As a result, exposure of the lower part of the bulbar conjunctiva and the cornea produces inflammatory changes which may lead to symblepharon and corneal ulceration. The lesions that characteristically affect the skin elsewhere involve that of the lids and, to a lesser extent, the conjunctiva.

The article is illustrated.

ZENTMAYER. [ARCH. OPHTH.]

POLLEN SURVEYS IN THE UNITED STATES: A CRITICAL REVIEW. PHILIP M. GOTTLIEB and ERICH URBACH, J. Lab. & Clin. Med. **28**:1426 (Sept.) 1943.

This is a review of the existing information regarding the distribution and times of pollination of the pollinosis-producing plants in the United States. Certain deficiencies in published data are pointed out. The authors suggest a correlated nationwide pollen survey based on simultaneous botanic observations, pollen counts and clinical investigations.

WILKINSON, Galveston, Texas.

SYSTEMIC ALLERGIC REACTION INDUCED BY YELLOW FEVER VACCINE. HARRY SWARTZ, J. Lab. & Clin. Med. **28**:1663 (Nov.) 1943.

The author presents a case of an atopic person who experienced a severe constitutional reaction immediately after injection of yellow fever vaccine. The patient was found to be sensitive to both egg and chicken. The author points out the necessity of knowing something of the constitution of the immunizing injection and of the subject to whom it is given.

WILKINSON, Galveston, Texas.

PEDICULOSIS CAPITIS AND CORNEAL LESIONS. A. HIRTENSTEIN, Brit. M. J. **2**:75 (July 17) 1943.

The author draws attention to the frequency of severe corneal lesions associated with pediculosis capitis. The patients were usually young children who were well fed and without apparent signs of general debility. The presence of pediculosis capitis was discovered by chance on the children's admission to the ward. There was no phthiriasis palpebrarum. Conjunctivitis was always present; corneal ulcers were noted in 14 of the 25 patients; marginal keratitis was observed in 8 patients; hypopyon ulcer in 1 patient and severe conjunctivitis without corneal lesions in 2 patients. Twenty patients were under 10 years of age, and 5 were older. T lesion was always unilateral, and the symptoms w burning pain followed by photophobia and mucopurul discharge. Bacteriologic examination showed nothi characteristic. The corneal lesions had no unusual fe tures. They were central or marginal, and sometim they spread around the corneal margin, forming a co plete ring of infiltration. The iris was congested, b there were no corneal deposits. The condition was u usual, as it showed little tendency to respond to routi treatment; in fact, it took a long time to heal. Trea ment of the eye consisted in phenolization of the ul and the use of atropine, mild protein silver, sulfapyr dine tablets and protein shock (injections of milk).

ARNOLD KNAPP. [ARCH. OPHTH.]

Surgery and Orthopedics

CASEIN IN THE LOCAL TREATMENT OF BURNS A WOUNDS. RAYMOND M. CURTIS and JOHN BREWER, Arch. Surg. **48**:130 (Feb.) 1944.

Searching for an agent for the treatment of bur that would be sterile, flexible, easily transported a inexpensive, the authors, after an exhaustive study the proteins available, found that the most satisfacto preparation was one made from casein. This is a ph phoprotein precipitated from milk by dilute acids and a readily obtainable by-product of milk. The soluti containing casein was used with a flexible transpare film in the local treatment of burns and wounds. Th method of treatment was applied in 240 cases of bur and wounds and showed a low incidence of infecti and a minimum of complications.

KAISER, Rochester, N. Y.

Teeth and Dentistry

THE ERUPTION OF THE DECIDUOUS TEETH. HARRY SANDLER, J. Pediat. **25**:140 (Aug.) 1944.

One thousand nine hundred and sixty-two white chi dren of fairly low economic status but under nutrition and medical supervision at the baby clinics of the Ne York City Department of Health provided material f the study of the eruption of deciduous teeth. The pr portion of children in whom each type of tooth migh be expected to erupt at each month of age was ca culated and smoothed curves plotted. The number teeth to be expected in a child's mouth at any age wa also calculated and plotted, together with a measure the variation to be expected.

It was found that no deciduous tooth had erupted i any child under 4 months of age and that all of th deciduous teeth had erupted in all of the children ov 34 months of age. The most usual order of eruption teeth was from earliest to latest: lower central incisor upper central incisors, upper lateral incisors, first molar (either upper or lower), cuspids (either upper or lower and second molars (either upper or lower).

Tests of correlation indicated that there is no charac teristic difference between boys and girls as to the age at which specific teeth erupt or in respect to the numbe of teeth which erupt at any age. The relationship be tween the weight of a child and the age of eruption i so slight as to be of no statistical significance. There i no significant difference in the time of eruption as be tween children of native and of foreign born mothers

AUTHOR'S SUMMARY.

Miscellaneous

AVISM. HUGH W. JOSEPHS, Bull. Johns Hopkins Hosp. **74**:295 (May) 1944.

Favism has been reported only twice in this country. Josephs reports a typical attack of hemolytic anemia which followed the eating of fava beans. After recovery a similar attack was induced experimentally. Although not grown in the United States, the fava bean is frequently imported from Mediterranean countries.

LYTTLE, Los Angeles.

IMUNOLOGIC AND TOXIC PROPERTIES OF CASEIN DIGEST AS PREPARED FOR PARENTERAL ADMINISTRATION. EDWARD C. HOPPS and JAMES A. CAMPBELL, J. Lab. & Clin. Med. **28**:1203 (July) 1943.

The importance of parenteral therapy to relieve conditions of protein deficiency is pointed out. Experiments are described by which two preparations of casein hydrolysate were tested for antigenic properties. The preparations tested were shown to be incapable of stimulating the production of antibodies; also, there were no reactions to preformed casein antibodies. Reactions of normal strips of smooth muscle to casein digests indicated the presence of some histamine-like substances, which, the authors suggest, may explain the frequently observed reactions of flushing, sensation of warmth and nausea in patients given a casein hydrolysate by vein. Furthermore, the authors point out that casein hydrolysates have a high hydrogen ion concentration, but that this does not appreciably affect the acid-base balance in persons with adequate hepatic function. Amino acids are conjugated at a rapid rate by the liver, but patients with poor hepatic function may be unable to conjugate parenterally administered amino acids at a sufficiently rapid rate to escape severe acidosis.

WILKINSON, Galveston, Texas.

A RAPID METHOD FOR DETERMINING THE SULFONAMIDE DRUG OF CHOICE. ROBERT J. ATWELL, RICHARD P. SEXTON and MARY A. POSTON, J. Lab. & Clin. Med. **28**:1620 (Oct.) 1943.

The authors describe a simple, quick method of testing the inhibiting effect in vitro of several sulfonamide compounds on the growth of various bacteria. The drug of choice is determined as soon as the control cultures are positive.

WILKINSON, Galveston, Texas.

BARBITURATE INTOXICATION AND PICROTOXIN TREATMENT. BENNETT W. BILLOW, J. Lab. & Clin. Med. **29**:265 (March) 1944.

A case of severe barbiturate poisoning in a 24 year old woman is reported. She had swallowed 3 Gm. of pentobarbital sodium. Metrazol and nikethamide were employed in the beginning of therapy, but with no clinical signs of improvement. Picrotoxin was then administered, a total of 135 mg. being given. Complete recovery resulted. The author emphasizes the increase in the incidence of barbiturate poisonings, intentional or accidental.

WILKINSON, Galveston, Texas.

Book Reviews

Control of Pain in Childbirth. By Clifford B. Lull and Robert A. Hingston. Price, $7.50. Pp. 356, with 10 plates and 100 other illustrations. Philadelphia: J. B. Lippincott Company, 1944.

From the introduction of chloroform into the field of obstetrics by Sir James Simpson until the present time obstetric anesthesia and analgesia have attracted wide interest. The advent of every new method of relieving pain has produced not only professional reports but also, with respect to some of the methods which have been advocated, articles in the popular press. Many of the latter group have been prepared by nonmedical writers who, at least in some instances, have had a small amount of information but much enthusiasm and occasionally some prejudice. Even obstetricians have been prone to favor the methods with which they were well acquainted and perhaps to undervalue others.

This monograph, by Drs. Lull and Hingston, will fill a useful place, and its appearance is timely. It covers the field of obstetric anesthesia and analgesia, giving adequate information as to indications and technic. In a number of instances, the authors have asked workers in clinics other than their own who have devoted especial attention to some form of relief of pain to contribute a section concerning that method. This insures a description by some one who has had sufficient experience with the method in question to explain it adequately. Drs. Lundy, McCormick, Tucker and Greenhill have discussed various phases of the field of relief of pain.

In part I is found a thorough exposition of the pelvic anatomy with particular reference to the spinal column and the sacrum. The nerve supply of the pelvic organs receives a discussion sufficiently long to bring out the points which are important from the standpoint of anesthesia. Two chapters deal with the physiopharmacology of anesthetic agents—general, local, spinal and caudal.

A brief history of relief of pain in childbirth follows in part II. In the remaining portion of part II the indications for various methods of relief of pain are discussed together with the technic of their use. The reader will find directions for the use of all the methods which have been proved useful sufficiently clear and detailed to enable him to use them intelligently. Anesthesia in cesarean section is well considered. Management of the third stage and the puerperium from the standpoint of relief of pain is included. The discussion of the selection of anesthetic agents for patients cared for during labor at home will be of use to many physicians who have this problem. The control of pain in maternal complications and the methods of resuscitation and drugs which may be used for that purpose occupy the terminal chapter.

The book is well illustrated. Many drawings, some of which are in color, add much to the descriptions of caudal and spinal anesthesia. Steps in technic are also made clear by many illustrations. The quality of the drawings as well as their number is to be commended.

The book may be warmly commended. One of the authors has had wide experience in the practice and teaching of obstetrics, and the other is one of the workers who developed the method of caudal anesthesia. Although both authors have been active in the adaptation of caudal anesthesia to obstetrics, both normal and operative, they have given an excellent review of the whole field of obstetric anesthesia and analgesia, including all methods which experience has shown to be of value. Their broadmindedness in asking workers in other clinics to prepare descriptions of technics other than those commonly used in their own institution indicates a desire to give their readers the fairest possible idea of different methods and of various points of view.

The book is a useful and practical one. Obstetricians and general practitioners whose work includes obstetrics will find it helpful, and even the anesthesiologist may find it of value.

The Hospital in Modern Society. By Arthur C. Bachmeyer, M.D., and Gerhard Hartman, Ph.D. Price, $5. Pp. 763. New York: Commonwealth Fund, 1943.

This book is a collection of material from recent periodical literature, each chapter containing several articles on a particular phase of hospital management and activity. The excellent selection indicates that extensive research was done by the editors, Dr. Arthur C. Bachmeyer and Dr. Gerhard Hartman, to include the best of writing in each field. The authors are recognized leaders in the hospital world, and the subjects are of practical import.

As a work of general instruction, it should prove of outstanding value. A busy hospital director will find it a handy reference, with valuable information from other hospitals to aid in supplying the broad overview necessary for competent administration. Board members interested in improving or enlarging hospital facilities may find much food for thought after studying material on similar departments in other hospitals, as described in the book. The volume can be read profitably, either as a whole or in part, by hospital department heads and by students of hospital management who desire added practical knowledge.

Interesting, as well as instructive, are the sections on the history of hospitals and their place in the social order.

Because of its wide range of subject matter, its practical ideas and its readability, "The Hospital in Modern Society" deserves a convenient place in the personal libraries of all who are interested in hospital management.

Clinical Urology. Vols. I and II. Second edition. By Oswald Swinney Lowsley, M.D., and Thomas J. Kirwin, M.D. Price, $10. Pp., vol. I, 974, with 220 illustrations; vol. II, 1,801, with 365 illustrations. Baltimore: The Williams & Wilkins Company, 1944.

A great deal of work has been given to the preparation of this edition.

The two volumes represent a complete treatise on diseases of the genitourinary system. Since the first edition, in 1940, many important developments in urologic diagnosis and treatment have occurred, and these are given careful consideration in this second edition. For example, the use of sulfonamide compounds and of penicillin in the treatment of infections of the urinary tract has been included. Recent developments in the treatment of cancer of the prostate are discussed.

The organization of the text is excellent, and the subjects and chapters are exceedingly well arranged, carefully balanced and well illustrated. The book is easily read. An extensive and authoritative bibliography has been appended to the chapters, and a complete index of the contents has been included in each volume. The surgical aspects of urology are given special attention. Although in a few isolated instances opinions may differ from those of the authors concerning the indications for certain types of operations, in the main the surgical procedures and the management of urologic diseases as presented are well established and standardized procedures which have been supplemented by the authors' extensive experience.

There are forty chapters, the first five of which take up the general diagnostic procedures in urology. Then follows a chapter on anesthesia. In the remaining chapters the organs of the urogenital system are considered as to their embryology, anatomy, anomalies, physiology, injuries and diseases. The pattern of each section is the same. The last two chapters deal with radium and Roentgen ray therapy of the genitourinary tract and with the therapy of dried blood plasma; these subjects are thoroughly discussed.

These two volumes should continue to have a wide field of usefulness in medicine. They are of value to the medical student as well as the general practitioner, besides being a good reference book for the trained urologist.

American Medical Practice in the Perspectives of a Century. By Bernhard J. Stern, Ph.D., Lecturer in Sociology, Columbia University; Visiting Professor of Sociology, Yale University. Price $1.50. Pp. 156, with index. New York: Commonwealth Fund, 1945.

Inasmuch as this is the first, as well as the basic, volume in a series of ten or more monographs to be published soon, the reviewer is of the opinion that a direct quotation from the foreword will serve best for purposes of orientation.

"The Committee on Medicine and the Changing Order was established by the Council of the New York Academy of Medicine in the winter of 1942 and began its work in February, 1943. This action was taken in the conviction that the medical profession is confronted by problems which require thorough study, and that the Academy had both the opportunity and the responsibility to contribute to the effective solution of these problems.

"The Council's instructions to the Committee were to review the nature, quality and direction of the economic and social changes that are taking place now and that are to be anticipated in the immediate future; to define in particular how these changes are likely to affect medicine in its various segments; to determine how the best elements in the science and art of medicine and its service to the public may be preserved, embodied and extended in whatever new social patterns may ultimately appear.

"The objectives of the Committee were defined as follows:

"To explore the possibilities and to formulate methods of maintaining and improving standards of quality in medical service, including medical research, medical education, the maintenance of health, both physical and mental, the prevention of disease, and the treatment of disease.

"To study the means of making available to larger groups of people and to the country as a whole the best known practice in preventive and curative medicine.

"To explore the possibilities and to formulate proposals of distributing these services not only to a larger number but also at a lower per capita cost than the present system permits.

"The Committee was composed of physicians, representatives of the allied professions of dentistry and nursing, and laymen.

"Being acutely aware of the need for orderly, systematic and objective data relative to the many aspects of medical education, medical research and medical service, the Committee early enlisted the coöperation of a number of experts in the composition of a series of monographs devoted to the major medical problems of today. These monographs are designed to provide the framework for an understanding of the current medical situation and its trends. Contemporary medicine is treated as a product of evolution. The monographs are historical in character; they are not chronological recitations, but show rather the reciprocal effects of medicine and the technical, social, economic and political changes that have taken place in American life.

"This treatment of the subject-matter brings to light not only the achievements but also the inadequacies in present-day medical practice—the uneven development of its different sectors, the prevailing incongruities and irrationalities, as well as the lag in the adjustment of medicine to the social needs of today. The monographs therefore offer not only a survey of the present situation but also an analysis of how it came to be and some indications as to future trends.

"In addition to the monographs, the Committee will issue a report representing the conclusion drawn from its deliberation and studies.

"The Committee has selected for the authors of its monographs recognized experts and has afforded them full freedom in their work. While, therefore, the monographs form an integral part of the Committee's studies, their publication does not necessarily imply the Committee's endorsement of statements of fact or opinion, which are entirely the responsibility of the authors."

This method of survey—the collection of all available evidence, the thorough and complete study of the facts and opinions encountered—is "the scientific method." If the problems are approached slowly and cautiously by these means, the consensus of the Committee should result in useful and valid conclusions for the benefit of members of the medical profession and the public.

This monograph is excellent; it is written with care and understanding. It is *must* reading for any one who is interested in the problem. It is hoped that the other monographs will be of the same high order of scholarship.

News and Comment

GENERAL NEWS

Fee for Taking Examination of American Board of Pediatrics.—After May 1, 1945 the fee for taking the examination of the American Board of Pediatrics will be $75.

PERSONAL NEWS

New Appointment for Dr. Albert D. Kaiser.—Dr. Albert D. Kaiser, 29 Buckingham Street, Rochester 7, N. Y., has accepted the position of health officer of Rochester, and the position of professor of child hygiene at the University of Rochester Medical School. Dr. Kaiser will assume his new duties on April 1, 1945.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof. S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Charles F. McKhann, University Hospital, Ann Arbor, Mich.
Canadian Committee:
Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.
Place: Boston. Time: Postponed indefinitely.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

Associação Paulista de Medicina, Section on Pediatrics

President: Dr. Vicente Laro.
First Secretary: Dr. Armando de Arruda Sampalo.
Second Secretary: Dr. Luiz Augusto de Toledo, Av. Brigadeiro Luiz Antonio 393, 1° Andar, São Paulo, Brazil.
Time: Twelfth of every month, 8:30 p. m.

British Paediatric Association

President: Prof. L. G. Parsons, 58 Calthorpe Rd., Five Ways, Birmingham.
Secretary: Dr. Donald Paterson, 27 Devonshire Pl., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. E. Gjørup, Dronning Louises Børnehospital, Copenhagen.

Nederlandische Vereeniging voor Kindergeneeskunde

President: Dr. J. H. G. Carstens, Servaasbolwerk 14ª, Utrecht.
Secretary: Dr. R. P. van de Kasteele, Laan van Poot 340, 's Gravenhage.
Place: Different places. Time: Three times a year.

Paediatrický Spolok na Slovensku

President: Dr. A. J. C. Chura, Lazaretská 11, Bratislava
Secretary: Dr. P. Rados, Lazaretská 6, Bratislava.
Place: Pediatric Clinic, University Bratislava. Time: Six times year.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. E. A. Cockayne, 98 Harley St., London W. 1, England.
Secretary: Dr. R. Lightwood, 86 Brook St., London W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. S. Rosenbaum, 26 Bialkstr., Tel Aviv
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

Sociedad Cubana de Pediatria

President: Dr. Angel A. Aballí Arellano, 17 No. 60 Vedado, Habana.
Secretary: Dr. Julio G. Cabrera Calderin, Hospital Mercedes L y 21 (Vedado), Box 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Mercedes Habana. Time: Last Wednesday of every month.

Sociedad Mexicana de Pediatria

President: Dr. Fernando López Clares, 12/a. Medellín 191, Mexico.
Secretary: Dr. Jesus Gómez Pagola, Versalles 6 Mexico.

Sociedad Venezolana de Puericultura y Pediatria

President: Dr. E. Santos Mendoza.
Secretary: Dr. P. Oropeza, Hospital de Niños, Caracas

Société de Pédiatrie de Paris

President: Dr. B. Weill-Hallé, 49 Avenue Raymond Poincaire, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue Pré aux Clercs Paris, France.
Place: Hôpital des Enfants Maladies, 49 Rue de Sèvres Time: 4:30 p. m., third Thursday of every month.

Uruguyan Society of Pediatrics

President: Dr. Jose Alberto Praderi, Eduardo Acevedo 1132, Montevideo.
Secretary: Dr. Hector Fossati, Juan D. Jackson 127 (Ap. 2), Montevideo.

NATIONAL

American Medical Association, Scientific Assembly, Section on Pediatrics

Chairman: Dr. John Aikman, 184 Alexander St., Rochester, N. Y.
Secretary: Dr. Gilbert J. Levy, 188 S. Bellevue Blvd., Memphis, Tenn.
In compliance with the request of the Office of Defense Transportation and in the interest of the national war effort a meeting will not be held in 1945.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Academy of Pediatrics

President: Dr. Joseph S. Wall, 1864 Wyoming Ave. N. W., Washington, D. C.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.

American Hospital Association, Children's Hospital Section

Chairman: Dr. Joelle C. Hiebert, 299 Main St., Lewiston, Maine.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly, Mass.

American Pediatric Society

President: Dr. James L. Gamble, 300 Longwood Ave., Boston.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis.

Canadian Society for the Study of Diseases of Children

President: Dr. R. R. Struthers, 906 Drummond Medical Bldg., Montreal.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, Hospital for Sick Children, Toronto.

Society for Pediatric Research

President: Dr. Edward M. Bridge, 219 Bryant St., Buffalo, N. Y.
Secretary: Dr. Mitchell I. Rubin, 1740 Bainbridge St., Philadelphia 46.

SECTIONAL

Intermountain Pediatric Society

President: Dr. Eugene Smith, 385–24th St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

New England Pediatric Society

President: Dr. Warren R. Sisson, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. James Marvin Baty, 1101 Beacon St., Brookline, Mass.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

North Pacific Pediatric Society

President: Dr. M. L. Bridgeman, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. C. G. Ashley, 833 S. W. 11th Ave., Portland, Ore.

Northwestern Pediatric Society

President: Dr. Arild E. Hansen, University of Minnesota, Minneapolis.
Secretary-Treasurer: Dr. Albert V. Stoesser, 205 W. University Hospital, Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Rocky Mountain Pediatric Society

President: Dr. G. R. Fisher, 23 E. Pikes Peak Ave., Colorado Springs, Colo.
Secretary: Dr. Joseph H. Lyday, 1850 Gilpin St., Denver.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. William Weston Jr., 1428 Lady St., Columbia, S. C.
Secretary: Dr. Angus McBryde, 604 W. Chapel Hill St., Durham, N. C.

STATE

Alabama Pediatric Society

President: Dr. Amas Gipson, 948 Forrest Ave., Gadsen.
Secretary-Treasurer: Dr. Ruth Berrey, 2021–6th Ave. N., Birmingham.

Arizona Pediatric Society

President: Dr. Vivian Tappan, San Clemente, Tucson.
Secretary: Dr. Hilda Kroeger, Arizona State Health Dept. (Maternal and Child Welfare Division), Phoenix.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. William C. Deamer, University of California Hospital, San Francisco.
Secretary: Dr. Charles W. Leach, 2000 Van Ness Ave., San Francisco.

Florida State Pediatric Society

President: Dr. Ludo Von Meysenbug, Box 3356, Daytona Beach.
Secretary: Dr. Robert Blessing, 409 Blount Bldg., Ft. Lauderdale.
Place: Concurrent with state association meeting at time of convention.

Georgia Pediatric Society

President: Dr. T. F. Davenport, 104 Ponce de Leon Ave. N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Edward T. Wakeman, 129 Whitney Ave., New Haven.
Secretary: Dr. Herman Yannet, Southbury Training School, Southbury.
Time: Three meetings a year.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. Craig D. Butler, 715 Lake St., Oak Park.
Secretary: Dr. A. J. Fletcher, 139 N. Vermilion, Danville.

Indiana State Pediatric Society

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Mathew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Mark L. Floyd, Children's Hospital, Iowa City.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport.

MEDICAL SOCIETY OF STATE OF NEW YORK, SECTION ON PEDIATRICS

Chairman: Dr. A. Clement Silverman, 608 E. Genesee St., Syracuse.
Secretary: Dr. Albert G. Davis, 307 Gas and Electric Bldg., Utica.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA, PEDIATRIC SECTION

Chairman: Dr. Elwood W. Stitzel, 403 Central Trust Bldg., Altoona, Pa.
Secretary: Dr. P. F. Lucchesi, Philadelphia Hospital, Philadelphia.

MICHIGAN STATE MEDICAL SOCIETY, PEDIATRIC SECTION

Chairman: Dr. Charles F. McKhann, University Hospital, Ann Arbor.
Secretary: Dr. Mark F. Osterlin, Central Michigan Children's Clinic, Traverse City.

MISSISSIPPI STATE PEDIATRIC SOCIETY

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

NEBRASKA PEDIATRIC SOCIETY

President: Dr. John L. Gedgoud, University of Nebraska Hospital, Omaha.
Secretary-Treasurer: Dr. Gerald C. O'Neil, 640 City National Bank Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

NEW HAMPSHIRE PEDIATRIC SOCIETY

President: Dr. MacLean J. Gill, 14 N. State St., Concord.
Secretary-Treasurer: Dr. Ursula G. Sanders, 46 Pleasant St., Concord.
Time: Twice yearly.

NORTH CAROLINA PEDIATRIC SOCIETY

President: Dr. Arthur H. London, 1105 W. Main St., Durham.
Secretary: Dr. Jay M. Arena, 604 W. Chapel Hill St., Durham.

OKLAHOMA STATE PEDIATRIC SOCIETY

President: Dr. Ben H. Nicholson, 301 N. W. 12th St., Oklahoma City.
Secretary: Dr. Luvern Hays, 108 W. 6th St., Tulsa.
Place: Oklahoma Club. Time: 6:30 p. m., fourth Friday of each alternate month from September to May, inclusive.

SOUTH CAROLINA PEDIATRIC SOCIETY

President: Dr. Lonita Boggs, 301 E. Coffee St., Greenville.
Secretary-Treasurer: Dr. Hilla Sheriff, Wade Hampton Office Bldg., Columbia.

TEXAS PEDIATRIC SOCIETY

President: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas.

VIRGINIA PEDIATRIC SOCIETY

President: Dr. L. E. Sutton Jr., Medical College Hospital, Richmond.
Secretary: Dr. Emily Gardner, 1100 W. Franklin, Richmond.

WEST VIRGINIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

President: Dr. Andrew Amick, 1021 Quarrier, Charleston.
Secretary: Dr. A. A. Shawkey, Professional B, Charleston.

LOCAL

ACADEMY OF MEDICINE OF CLEVELAND PEDIATRIC SECTION

Chairman: Dr. J. W. Epstein, 10515 Carnegie, Cleveland.
Secretary: Dr. Virginia Barker, City Hospital, Cleveland.
Place: Cleveland Medical Library Bldg. Time: tober, December, February and April.

ACADEMY OF MEDICINE, TORONTO, SECTION OF PEDIATRICS

President: Dr. I. Nelles Silverthorne, 170 St. G St., Toronto, Canada.
Secretary: Dr. G. P. Hamblin, 2333 Bloor St. Toronto, Canada.

BRONX PEDIATRIC SOCIETY

President: Dr. Jacob H. Turner, 121 W. 197th New York.
Secretary: Dr. Harry S. Pizer, 1726 Davidson A New York.
Place: Concourse Plaza Hotel, 161st St. and G Concourse. Time: Second Wednesday of each m except June, July, August and September.

BROOKLYN ACADEMY OF PEDIATRICS

President: Dr. Kenneth G. Jennings, 159 Midwood Brooklyn.
Secretary: Dr. Joseph Battaglia, 713 Bushwick A Brooklyn.
Place: Granada Hotel. Time: Fourth Wednesday each month except May, June, July, August September.

BUFFALO PEDIATRIC SOCIETY

President: Dr. A. Wilmot Jacobsen, 187 Bryant Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest A Buffalo, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8 p. m., first Monday of each month from Septe to June.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Edward J. Wynkoop, 501 James Syracuse.
Secretary Dr. Frank J. Williams, 58 S. Swan Albany.
Places: Various cities in New York. Time: T Tuesday of April and September.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Morley D. McNeal, 2 N. Sheridan Highland Park, Ill.
Secretary: Dr. Henry G. Poncher, 1819 W. Polk Chicago.
Place: Children's Memorial Hospital, 710 Fulle Ave. Time: Third Tuesday of each month, Oct to May, inclusive.

Cincinnati Pediatric Society

dent: Dr. Lloyd K. Felter, 3144 Jefferson Ave., cinnati.
tary: Dr. T. Selkirk, 3530 Reading Rd., Cincinnati.
: Children's Hospital, Elland Ave., Cincinnati.
me: On call.

Dallas Pediatric Society

dent: Dr. Robert A. Knox, 3701 Fairmount Ave., llas 4, Texas.
tary-Treasurer: Dr. Gladys J. Fashena, 4585 Bel- t Pl., Dallas 4, Texas.
: Children's Medical Center. Time: 1 p. m., second d fourth Saturdays of each month.

Detroit Pediatric Society

dent: Dr. John J. Pollack, 650 Maccabees Bldg., troit, Mich.
tary: Dr. Philip J. Howard, 2799 W. Grand Blvd., troit, Mich.
: Wayne County Medical Society. Time: 8:30 m., first Wednesday of each month from October June, inclusive.

Fulton County Medical Society, Pediatrics Section (Atlanta, Ga.)

rman: Dr. Don F. Cathcart, 478 Peachtree St. E., Atlanta.
retary: Dr. Harry Lange, 478 Peachtree St. N. E., lanta.
e: Academy of Medicine, 38 Prescott St. N. E.
me: Second Thursday of each month from October April, 8 p. m.

Houston Pediatric Society

sident: Dr. Raymond Cohen, 2300 Caroline St., ouston, Texas.
retary: Dr. Betty Moody, 526 Richmond Rd., ouston, Texas.
e: College Inn, Houston. Time: Fourth Monday each month.

Kansas City (Missouri) Pediatric Society

sident: Dr. Edwin H. Schorer, 1103 Grand Ave., ansas City.
retary: Dr. H. E. Petersen, Kirkpatrick Bldg., St. seph, Mo.
e: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

sident: Dr. Milo B. Brooks, 1033 Gayley Ave., West os Angeles.
retary-Treasurer: Dr. Elena Boder, 1830½ Lucille ve., Los Angeles 26.
e: Los Angeles County Medical Headquarters, 1925 ilshire Blvd. Time: Second Monday of February, pril, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

sident: Dr. Abraham M. Litvak, 1145 Eastern Park- way, Brooklyn.
retary: Dr. Harold Levy, 750 St. Marks Ave., rooklyn.
e: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., ourth Monday of each month, October to April, clusive.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. Meyeron Coe, 217-02-91st Ave., Queens Village, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144-38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry A. Spigel, 2647 Connecticut Ave., Washington, D. C.
Secretary-Treasurer: Dr. Perry W. Gard, 2520 Woodley Rd., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of every month.

Memphis Pediatric Society

President: Dr. F. T. Mitchell, 376 S. Bellevue Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Harry Jacobson, 1193 Madison Ave., Memphis, Tenn.
Place: John Gaston Hospital. Time: Quarterly.

Milwaukee Pediatric Society

President: Dr. M. G. Peterman, 411 E. Mason St., Milwaukee 2.
Secretary-Treasurer: Dr. B. J. Malnekoff, 3213 N. Marietta Ave., Milwaukee.
Place: Milwaukee Athletic Club. Time: Second Wednesday of each alternate month, beginning with February.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Howard Craig, 175 E. 79th St., New York.
Secretary: Dr. Alfred E. Fischer, 73 E. 90th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northern California Pediatric Society

President: Dr. John Mott Rector, 2000 Van Ness Ave., San Francisco.
Secretary: Dr. George Bates, 490 Post St., San Francisco 2.
Time: Four times yearly.

Oklahoma City Pediatric Society

President: Dr. William M. Taylor, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 625 N. W. 10th St., Oklahoma City.
Place: Oklahoma Club. Time: Third Thursday of each month.

Philadelphia Pediatric Society

President: Dr. Carl Fischer, Greene and Coulter Sts., Germantown, Philadelphia.
Secretary: Dr. Sherman Little, 1740 Bainbridge St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday in January, March, May and November.

Pittsburgh Pediatric Society

President: Dr. John D. Sturgeon Jr., 22 N. Gallatin Ave., Uniontown, Pa.
Secretary-Treasurer: Dr. C. J. Stoecklein, Medical Arts Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate month from October to June, inclusive.

Richmond Pediatric Society

President: Dr. Stanley Meade, 913 Floyd Ave., Richmond, Va.
Secretary-Treasurer: Dr. Louise Galvin, 214 S. Boulevard, Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: 8 p. m., third Thursday of each month, except June, July and August.

Rochester Pediatric Society

President: Dr. Herbert Soule, 122 Rutgers St., Rochester, N. Y.
Secretary-Treasurer: Dr. Jerome Glaser, 300 S. Goodman St., Rochester, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Third Friday of each month from October to May.

St. Louis Pediatric Society

President: Dr. Peter G. Danis, 634 N. Grand Ave., St. Louis 3.
Secretary-Treasurer: Dr. H. Ewing Wachter, 3720 Washington Ave., St. Louis.
Place: St. Louis Medical Society Bldg. Time: Third Thursday of each month from November to June.

Seattle Pediatric Society

President: Dr. Frederick B. Joy, Stimson Bldg., S
Secretary: Dr. Sherod M. Billington, Medical D Bldg., Seattle.
Place: College Club. Time: Third Friday of month from September to June at 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Jeanette Harrison, 1136 W. 6th Los Angeles.
Secretary: Dr. Henry F. Gallagher, 1930 Wi Blvd., Los Angeles.
Place: Jonathan Club of Los Angeles. Time: Wednesday in January, March, May, September November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Campbell Harvey, 35 W. Huron Pontiac, Mich.
Secretary: Dr. Harry A. Towsley, University of M igan, Department of Pediatrics and Communic Diseases, Ann Arbor, Mich.

Westchester County Medical Society, Pediatric Section (New York)

President: Dr. John B. Ahouse, 27 Ludlow St., Yon N. Y.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 High Rd., Rye, N. Y.
Place: Grasslands Hospital, Valhalla, N. Y. T Third Thursday in October, December, February April.

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